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EDITORIAL

APPENDICITIS MORTALITY

THE medical profession concedes that the mortality of appendicitis is far too high and that the question presents, as Royster says, a real tragedy because the deaths may be largely prevented if the initial treatment of the disease is correct.

The present-day statistics mean little or nothing and are in need of revision if the profession is to learn just what are the most important causes of this unnecessarily high mortality.

Without doubt, the delay of the patient in seeking medical relief, the medical treatment of the attack, the use of purgatives and of morphine and the mistakes in diagnosis are the main causes for this unnecessarily high mortality, but there are other causes which are important and upon which there is little light; namely, the use of the wrong type of operation performed at the wrong time and the wrong treatment of general peritonitis. These factors are great contributing causes of the present mortality and they have not been stressed sufficiently.

The reported grouped statistics mean little because the mortality rates vary with the number of chronic and acute clean cases included in a given series. It has been found that some surgeons, in their anxiety to show a remarkably low record for their series, include the secondary appendectomies performed during the course of other abdominal operations.

In revising the statistics all chronic and all acute simple cases in which the pathology still remains within the coats of the appendix should be discarded and our attention

centered only upon those in which the pathology no longer remains within the appendix. These cases may be termed appendicitis with complications.

If this plan were followed, the mortality would be found to be very high, but we would be in a position to decide, by comparison, the merits of the delayed operation over the immediate operation, the radical treatment—appendectomy, over the conservative—drainage, in the presence of abscess formation and the merits of the different treatments used in the forms of spreading or general peritonitis.

I venture the opinion that the conservative methods of treatment for these grave conditions would then show a far lower mortality than the radical ones advocated by some excellent surgeons who would hesitate to employ such radical measures in the treatment of spreading infectious processes in other parts of the body.

DONALD GUTHRIE.

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LIVER ABSCESS

PART I

AMEBIC ABSCESS
ANALYSIS OF 73 CASES*

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UP to the beginning of the present century relatively little was known concerning the etiology of solitary abscesses of the liver. Even though in 1891 Councilman and Lafleur[1] in their extensive and complete clinical and pathological investigations, definitely showed that amebae could produce solitary and multiple liver abscesses, it was not until Sir Leonard Rogers[2] in 1902 established that solitary "tropical" abscess was invariably the result of amebic infection. In a consideration of liver abscess, it is important from a prognostic and therapeutic standpoint to differentiate amebic from pyogenic abscesses. A rational consideration of liver abscess is not possible unless this differentiation is made. Such differentiation is imperative, because in amebic hepatic abscess the prognosis is good and the treatment relatively simple, whereas in pyogenic abscess the prognosis is extremely grave and the treatment more difficult.

The present report is based upon a study of 102 cases of liver abscess admitted to the Charity Hospital and the Touro Infirmary in New Orleans during the six year period, 1928 to 1933, inclusive. Of this number, 73 (71.5 per cent) were amebic abscesses and 29 (28.4 per cent) were pyogenic (Fig. 1). This latter group will be considered in detail in a subsequent publication. Many of the Charity Hospital cases were included in Gessner's[3] report.

ETIOLOGY

Ballingall,[4] in 1818, observed in 35 autopsies on patients with dysentery, 4 cases with liver abscess, and although amebae were not found, the causal relationship between the dysentery and the liver abscess was considered. Koch,[5] in 1883, found amebae in colonic ulcers of 5 patients dying of dysentery. Two of these had abscesses of the liver. In one of these amebae were found in the capillaries and tissues adjoining the abscess. Kartulis,[6] in 1889, stated that liver abscesses associated with dysentery were probably amebic in origin. He believed that the amebae were carried to the liver by means of the portal vein. In 1890 Osler[7] found amebae in a liver abscess of a patient operated upon and in whom subsequently the amebae were found in the stools. In Councilman and Lafleur's[1] 15 cases of amebic dysentery, 6 abscesses of the liver were found, of which 4 were multiple. Sir Leonard Rogers,[2] in 1902, definitely established that active amebae were constantly found in the wall of tropical abscesses of the liver, although they were frequently not present in the "pus." The ameba was the only organism constantly found in such abscesses. In 90 per cent

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of his cases, amebic dysentery definitely preceded the lesion in the liver and in the remaining 10 per cent such an antecedent

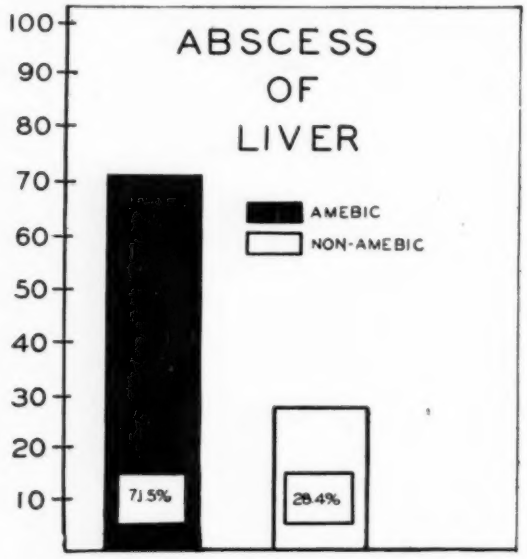

Fig. 1. Graphic representation showing incidence of amebic and non-amebic abscesses of the liver in a total of 102 cases of liver abscess.

infection could not be excluded. Rogers[2] also showed, that in the majority of instances pyogenic organisms were absent from these abscesses when they were first opened.

INCIDENCE

The incidence of abscess formation in amebic dysentery varies considerably according to the various reports. In a group of 4392 fatal cases collected from the literature[4,5,8–23] there were 1664 (37.9 per cent) cases of amebic abscess of the liver (Table 1). The highest incidence reported was by Smith,[21] who in 45 fatal cases of amebic dysentery found 38 cases of amebic liver abscess (84.4 per cent). Almost as high was a series of 699 cases reported by Edwards and Waterman[19] with 504 cases of amebic liver abscess, an incidence of 72.1 per cent. The incidence of amebic abscess occurring in all cases of amebic dysentery varies considerably according to different reports and apparently depends upon whether the cases of amebic dysentery were ambulatory or not. Lacaze and Melnotte[24] observed 95 (1.9 per cent) cases of amebic hepatic abscess among 5000 cases of amebic dysentery. Murray[25] states that the incidence of dysentery was 1.8 per cent of all the admissions in a Korean hospital. Over half of these had liver abscess, an incidence of approximately 0.9 per cent.

Table 1
Incidence of amebic liver abscess in fatal cases of amebic dysentery

Author	Amebic Dysentery	Amebic Abscess
Ballingall[4]	35	4
Annesley[8]	51	26
Waring[9]	259	69
Eyre[10]	118	27
Ranking[11]	140	41
Moore[12]	494	90
Macpherson[13]	293	46
Chuckerbutty[14]	39	3
Catteloup[15]	240	47
Mouret[16]	761	133
Parkes[17]	61	13
Kartulis[6]	500	300
Zancarol[18]	444	262
Edwards and Waterman[19]	699	504
Craig[20]	74	25
Smith[21]	45	38
Strong[22]	79	14
Craig[23]	60	22
Total	4392	1664 (37.9%)

Buchanan[26] found in India that the ratio of hepatic abscess to amebic dysentery was 1 to 628 in the natives and 1 to 18 in the Europeans. In 1000 cases of positive amebic infection in Peiping Tao[27] found liver abscess in 18. Futcher[28] reported 119 cases of amebic dysentery from the Johns Hopkins Hospital among which there were 27 cases of hepatic abscess, an incidence of 22.6 per cent. In the Charity Hospital group of our series there were 59 amebic abscesses of the liver among 388 cases of amebic dysentery, an incidence of 15.2 per cent. The high incidence in the Charity Hospital series and that of Futcher[28] can undoubtedly be explained on the basis that probably only the severer cases of amebiasis were admitted to the institutions, whereas the majority of the cases were treated ambulatory and were,

therefore, not included in the series. During the period of 1928 to 1933 there were 253,829 total admissions to the Charity Hospital, among which there were 84 amebic and pyogenic abscesses of the liver, an incidence of 0.033 per cent. In this series there were 59 amebic abscesses of the liver, an incidence of 0.023 per cent of the total admissions. During this same period there are 574 patients diagnosed as having liver disease, of which 14.6 per cent were abscesses and 10.2 per cent were amebic abscesses. Eolian[29] found that of 3115 hospital surgical patients there were 8 cases of amebic abscess of the liver and 1 amebic abscess of the subdiaphragmatic space, an incidence of 0.28 per cent. In the reported cases the greatest age incidence of amebic hepatic abscess has been in young adults, whereas it is lowest in children and elderly individuals. Waring[9] reports that 49.37 per cent of his patients were between twenty and thirty years of age. According to Ludlow[30] the majority of patients were from twenty to forty years of age. In Young's[31] series of 20 cases the oldest was fifty-two, the youngest seventeen, and the average thirty-five years. In the 73 cases in our series the youngest was eight and the oldest seventy, the average age being forty-four years. In contrast to the reported series, the majority of our patients, 54.7 per cent, were forty years or older, whereas only 45.2 per cent were younger than forty, and 4 patients were over sixty. The reason the highest incidence is in young adults in the series reported from the tropics is probably because amebiasis is more prevalent in the tropics and the span of life shorter than in temperate zones.

Males are much more susceptible to amebic abscess than females. The reasons which have been advanced by Rolleston[32] for this increased incidence among males are: (1) alcoholism, which predisposes to hepatitis, and is more frequent in males; and (2) there is greater likelihood of trauma in the male. Whereas the male is more predisposed to the development of amebic abscess, the incidence varies from 1 to 63 in the series reported by Thurston[33] to approximately 1 to 5 in those reported by Boland[34] and by Young[31] (Table II). In our own series there were 8 females and 65 males, a ratio of 1 to 8. Because Young's[31] and Boland's[34] series so closely parallel ours as regards the sex incidence, it is suggested that the predisposition to amebic liver abscess in males is less marked in the Southern States than it is in tropical countries.

TABLE II
SEX INCIDENCE IN AMEBIC LIVER ABSCESS

Author	Male	Female
Young[31]	17	3
Ludlow[30]	92	8
Giggam[42]	24	1
Thurston[33]	63	1
Boland[34]	5	1
Chen et al.[37]	46	2
Waring[44]	291	9
Manson-Bahr[43]	39	6
Authors	65	8
Total	642	39
Ratio	16.4 to 1	

The reports from India demonstrate that Europeans are more subject to amebic abscess formation than are natives. In Buchanan's[26] series there were 441 cases among the white inhabitants and 127 among the natives. Ludlow[30] also found that the whites were more susceptible to the development of amebic abscess than were the natives. In the Canal Zone Herrick[35] found that the white employees from the United States were more susceptible to amebic liver abscess than the white employees from Europe, who in turn were more susceptible than the natives. Among the white employees from the United States there was one case in every 1178, and among the whites from Europe there was one case in 2240, whereas among the black laborers there was one case in every 3722. In Young's[31] 20 cases there were 16 white and 4 colored. In our own series there were 44 whites and 29 colored.

As there are no colored patients admitted to the Touro Infirmary, only the Charity Hospital group of our series can be considered. In this group of 59 cases there were 30 white and 29 colored patients, showing an equal distribution between the two races. As the admissions for the two races were about equal, this incidence is absolute and not relative. The relatively greater proportion of colored patients admitted to the Charity Hospital, because there is only one other institution in New Orleans admitting negroes, accounts for the predominance of colored patients in our series as compared with the other reported series. Another possible reason is that the white patient is more apt to have antiamebic therapy before admission to the hospital, which decreases the incidence of liver abscess.

Aside from age and sex there are other predisposing factors for the development of an amebic liver abscess. The principal one of these is alcoholism. Rogers[2] obtained a positive history of the use of alcohol in 24 of 44 cases. Rolleston[32] believes that not only the excessive ingestion, but also the moderate use of alcohol predisposes to amebic hepatitis, as evidenced by increased incidence of abscess among the natives who are not total abstrainers. Sandwith[36] states that hepatic abscess is found only in those natives who are addicted to the use of alcohol. Waring[9] in 40 cases among native Indians found only 13 (32.5 per cent) were sober and temperate, whereas 27 (67.5 per cent) were intemperate. Chen, Gorder, and Yuan[37] state that 21 of 31 cases admitted the use of alcohol. Eolian,[29] on the other hand, does not feel that alcohol is a predisposing factor, because in the natives of Armenia who consume large quantities of alcohol the incidence of amebic abscess of the liver is less than in the Europeans who consume less alcohol. Unfortunately, in our own series we can draw no conclusions concerning the part that alcoholism plays, because in the amebic abscess group in only 4 cases was any statement made concerning the use of alcohol. In 2 of these alcoholism was present, whereas in the other 2 the patients were total abstainers. Megaw[38] found in 170 cases of amebic liver abscess that 119 (70 per cent) habitually took alcohol and 51 (30 per cent) did not. According to Rogers[2] there is no seasonal variation in amebic hepatic abscess. In 236 cases in which the seasonal incidence was determined he obtained the following: from January to March, 59; April to June, 58; July to September, 58; October to December, 61 cases. On the other hand, Rolleston[32] opines that an important factor is the sudden variation in temperature and that amebic hepatitis is more apt to occur at the onset of the cold season. Schrumpf-Pierron[39] believes that in Egypt the most important cause of the recurrence of amebic hepatitis is the hot winds which suddenly occur.

PATHOGENESIS OF AMEBIC HEPATIC ABSCESS

Due to the investigations of Councilman and Lafleur[1] and Rogers[2] the cause of amebic hepatitis has been definitely established as the result of invasion of the liver by the amebae. Kartulis[6] prior to these investigators thought that the amebae played at least a passive rôle in transporting pyogenic microorganisms to the liver and by rupturing the capillaries of the portal system by their active movements. At the present time the majority of authors are agreed that the amebae are carried to the liver by the portal system. Councilman and Lafleur[1] stated, "it is far more likely that amebae reached the liver by another route than through the blood and lymphatic vessels. This is by means of the abdominal cavity." This contention was based upon the observations that the most frequent sites of amebic abscess of the liver in their series were in the periphery either on the diaphragmatic or the colonic surfaces and that in one of their cases of liver abscess there was concomitant amebic peritonitis in the right upper abdominal quadrant. Rogers,[2,47,48] on the other hand, contends that the route of extension is

through the portal system. His explanation is as follows[47]:

Taking all these facts into consideration I suggest that in the slight or latent amebic colitis from which large amebic abscesses usually result, the comparatively few amebae reaching the liver become entangled in blood clots in the small interlobular veins, producing congestion of the organ, but that the vast majority of them undergo degeneration within the vessels, not being able to escape through the vessel-walls without some previous damage to their coats, in which case only what I have called a presuppurative amebic hepatitis results. If septic organisms are also present, softening of the vein walls and multiple small abscesses may occur with slight bowel lesions, as I have already shown, but these are exceptional cases. When, however, the amebae happen to reach some part of the liver in such numbers as to cause clotting of several contiguous small veins so as to interfere with the blood supply sufficiently to produce a small focal necrosis with consequent softening of the walls of the veins allowing escape of the amebae into the soft liver tissue, the commencement of an abscess results. The organisms then spread along the open veins producing further clotting and softening, as I have shown occurs at the margin of an actively spreading ragged walled abscess, with further breaking down of the liver substance and extension of the cavity, and this process continues in concentric circles until, if the patient survives long enough, a fibrous capsule develops, when the abscess only increases by distention of the cavity by continued formation of pus from its walls, but without further destruction of liver tissue. Thus I conclude that almost all forms of amebic liver abscess are due to pure amebic infections from the bowel through the portal veins with variable numbers of amebae, while it is also easy to understand that whether an amebic hepatitis goes on to suppuration or not may often be in the balance, so that certain predisposing causes, such as chills or alcohol, may easily turn the scale and bring about suppuration, which early diagnosis and suitable treatment may prevent.

Granted that the invasion of the liver in amebic hepatitis occurs by way of the portal stream, it is rather difficult to understand why amebic abscesses are primarily single, whereas the usual metastatic abscesses following a pylephlebitis are multiple. Craig[49] believes that amebic abscesses are more apt to be solitary than multiple even though carried through the portal stream, because whenever an ameba gains entrance to the blood stream, it is accidental and in all probability the number of amebae which reach the liver is relatively small as compared with the number of pyogenic microorganisms passing to the liver in cases of pylephlebitis, because in pyogenic infections invasion of the blood stream is the usual finding. We are inclined to believe from our experience that the involvement of the liver occurs through the portal system in all cases.

The majority of amebic abscesses of the liver are sterile. Fifty per cent of Craig's[20] cases were secondarily infected. In only 46 of our 73 cases of amebic abscess was any statement made concerning the bacteriologic examination of the pus obtained from the abscess. In 5 (10.8 per cent) secondary infection was present, whereas the remaining 41 (89 per cent) were sterile. The organisms which were found were staphylococci in 2 cases; streptococci in 1 case; staphylococci and streptococci combined in 1 case, and B. coli in 1 case. In a series of collected cases* together with our own cases there were 328 (83.9 per cent) of 386 which were sterile (Table III).

Table III
Incidence of Sterility of Amebic Abscess of Liver

Author	No. of Cases	Sterile
Kruse and Pasquale[52]	15	4
Rogers[47]	87	75
Futcher[28]	120	104
Okinschewitsch[50]	58	55
Manson-Bahr[43]	45	41
Talbot[54]	15	8
Authors	46	41
Total	386	328 (83.9%)

* See ref. 28, 43, 47, 50, 52, 54.

Councilman and Lafleur,[1] Rogers,[2] and Manson-Bahr[43] have emphasized the fact that active motile amebae are almost

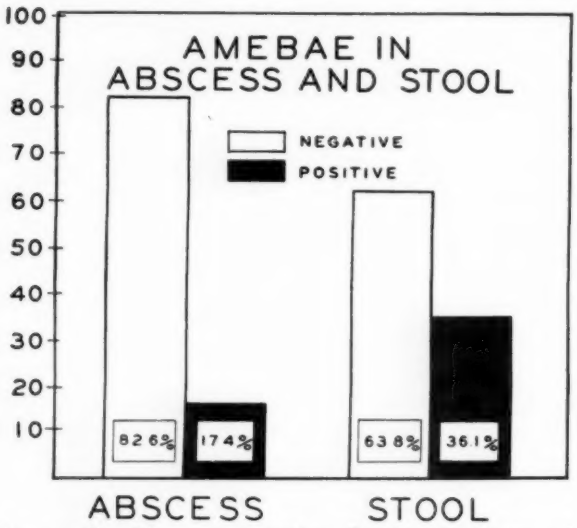

FIG. 2. Graphic representation showing incidence of positive findings of amebae in hepatic abscesses and in stool of patients with amebic hepatic abscess.

invariably found in small, early, acute abscesses, whereas they are seldom present in the pus of large abscesses. Although amebae are usually not found in the pus of amebic abscesses when first opened, they can generally be revealed in scrapings from the wall of the abscess. Justi[51] states that whereas the pus at the time of drainage contains no amebae, usually three or four days after either aspiration or drainage many amebae are present in the pus. This is probably due to the squeezing of the amebae into the abscess cavity caused by contraction of the abscess wall. In 46 cases in our series in which an investigation was made, the amebae were found in 8 instances (17.4 per cent) (Fig. 2). In 424 cases collected from the literature* together with our cases there were 234 (55.2 per cent) in which amebae were found in the abscess (Table IV). The high incidence of positive findings in Roger's[47] and Futcher's series[28] and that of Chen et al.[37] may be accounted for by the fact that scraping of the abscess wall was generally performed. The low incidence of positive findings (17.4 per cent) in our own series is due to the infrequency of performing this procedure. Obviously in those cases in which conservative treatment is used the finding of amebae will be even less.

* See ref. 25, 27, 28, 21, 42, 47, 50, 52, 53, 54.

TABLE IV
PRESENCE OF AMEBAE IN AMEBIC LIVER ABSCESS

Author	No. of Cases	Amebae Found
Rogers[47]	37	35
Futcher[28]	120	119
Chen et al.[37]	40	34
Young[31]	15	7
Okinschewitsch[50]	58	7
Kruse and Pasquale[52]	15	6
Candler[52]	12	3
Murray[25]	26	4
Talbot[54]	15	3
Giggam et al.[42]	25	0
Tao[27]	15	8
Authors	46	8
Total	424	234 (55.2%)

MORBID ANATOMY

In the majority of instances the right lobe of the liver is involved more frequently than the left. According to Snyder,[40] 75 per cent of the amebic hepatic abscesses occur in the right lobe. Moynihan[41] states that 71 per cent occur in the right and 16 per cent in the left. Waring[9] found the right lobe involved in 67 per cent, the left lobe in 6.6 per cent, and both lobes in 14.5 per cent. In Rouis's[45] series the lesion was in the right lobe in 78.6 per cent, the left in 16.8 per cent, and the Spigelian lobe in 4.6 per cent. In our own series there were 52 cases (91.2 per cent) in which the right lobe was involved, 2 (3.5 per cent) in which the left was involved, 2 (3.5 per cent) in which the median lobe was involved, and only 1 (1.7 per cent) in which both lobes were involved. We were able to collect from the literature 437 cases in which the location of the abscess was stated. To this we can add 57 of our own, making a total of 494 cases. If the autopsy series[1,20,44] are separated from the clinical series,[30,37,42,43]

interesting results are obtained. There were 219 cases in the autopsy series with the following localization: right lobe, 178 (81.2 per cent), left lobe 18 (8.2 per cent), and bilateral, 23 (10.5 per cent). In the clinical series were 275 cases with the localization as follows: right lobe, 254 cases (92.3 per cent), left lobe, 13 cases (4.7 per cent), median lobe, 2 cases (0.7 per cent), bilateral, 6 cases (2.1 per cent) (Table v).

TABLE V
SITUATION OF ABSCESS

Autopsy Cases

Author	Right	Left	Median	Bilateral
Councilman and Lafleur[1]	4	.....	.....	2
Craig[20]	11	2	.....	11
Waring[44]	163	16	.....	10
Total	178 (81.2%)	18 (8.2%)	.....	23 (10.5%)

Clinical Cases

Author	Right	Left	Median	Bilateral
Ludlow[30]	93	2	.....	5
Giggam[42]	22	3		
Chen et al.[37]	47	1		
Manson-Bahr[43]	40	5		
Authors	52	2	2	1
Total	254 (92.3%)	13 (4.7%)	2 (0.7%)	6 (2.1%)

The greatest differences in the two series are that the highest incidence of right lobe abscesses is in the clinical series and the highest incidence of bilateral involvement is in the autopsy series. This is probably due to several factors: (1) those cases coming to autopsy are the more severe ones with more extensive involvement of the liver; (2) in the clinical series many small abscesses resolve without being recognized, whereas the thorough postmortem examination will reveal even these small lesions. Davidson[46] found single abscess in 75 per cent of cases, 2 abscesses in 11 per cent, and more than 2 in 14 per cent. Moynihan[41] reports 70 per cent of his cases were single abscesses, 15 per cent multiple, and in 15 per cent, 2 abscesses were found. In our series of 73 cases of hepatic amebic abscess there were 45 (61.6 per cent) in which the abscess was single, 6 (8.2 per cent) with multiple abscesses, and 22 (30.1 per cent) in which the number was not stated. In all probability many of the cases in which it was not stated whether the abscesses were multiple or single, the abscesses were actually multiple. Including our own series there were 341 cases[1,23,28,44] (Table VI) in which

TABLE VI
INCIDENCE OF SINGLE AND MULTIPLE ABSCESSES

Author	Single	Multiple
Futcher[28]	10	8
Councilman and Lafleur[1]	2	4
Craig[23]	9	14
Waring[44]	177	66
Authors	45	6
Total	243 (71.2%)	98 (28.7%)

the number of abscesses was stated; 243 (71.2 per cent) were single and 98 (28.7 per cent) multiple.

According to Councilman and Lafleur,[1] amebic abscesses of the liver may be divided into three types: (1) the small, which are very acute, (2) the larger abscesses with partially fibrous walls, and (3) the chronic with hard, dense fibrous walls. There is considerable difference in these abscesses both histologically and as regards the number of amebae that they contain. Rogers[47] believes that the primary lesion in the liver is a pin-head sized abscess resulting from clotting in an interlobular branch of the portal vein with associated intracellular infiltration and softening of the walls of the vein. As a result of the clotting and inflammatory processes there occurs invasion of the liver parenchyma by active amebae. Rogers[47] is of the opinion that when multiple abscesses occur as a result of amebic infection it is either due to an associated pyogenic infection or to massive infection of the liver with amebae alone and that the lesion primarily is an anemic necrosis. According to Craig[49] the amebae play a cytolytic and a mechanical

rôle in the development of the abscess. He[55] showed that alcoholic extracts of amebae exert a lytic effect on intestinal mucosa. In addition to this action he believes[49] with Rogers[47] that amebae can produce anemic necrosis in the liver by mechanically plugging portal radicals. Rogers[47] states that rarely an intermediate abscess may be seen between the small acute abscess and the large chronic abscess. These are large ragged cavities without well-defined walls, which appear to be a number of coalescing abscesses. In these cases it is apparent that the process is extending along the vein from the central cavity. The condition is really a more extensive process of the development of the small multiple abscesses. The solitary abscess cases are usually associated with relatively small intestinal ulcers, whereas the multiple liver abscesses are more apt to develop following extensive ulceration of the bowel in which there is flooding of the portal vein with many amebae. If the process continues long enough, a fibrous tissue capsule will be formed, walling off the abscess. Rogers[47] believes that following this the abscess increases only by the continued formation of the pus from its walls, and without further destruction of the liver tissue. He emphasizes the importances of this limiting fibrous membrane and believes that further destruction of the liver occurs only when there is a complicating secondary infection. Such extension occurs because of the permeation of the abscess wall by pyogenic microorganisms which possess more invasive characteristics than do amebae. This frequently results in multiple secondary abscesses. Whereas the majority of amebic liver abscesses are sterile at the time of drainage, they rapidly become infected following open operation. Rogers[2] found that a few days after open drainage innumerable cocci and other bacteria were invariably present, which he believes is more apt to occur in the tropics than elsewhere. According to Councilman and Lafleur[1] contents of the early small abscesses are viscid, glary, and semitransparent, whereas in the older abscesses the contents are less viscid and are mixed with blood, which is responsible for the characteristic chocolate sauce appearance. The finding of chocolate-brown pus is so characteristic that Constantini[56] and Manson-Bahr[43] believe that one is justified in making a positive diagnosis of amebic infection even though the amebae are not found. The contents of the abscess characteristically are not purulent and contain few or no pus cells. Small solid particles of liver tissue which have resisted destruction and active amebae are found in the contents of newly formed abscesses. In large abscesses cellular detritus and blood are present, but amebae are usually absent. However, the amebae are almost invariably present in the wall of the abscess. As a result of the increasing size of the abscess, especially in the chronic form, there is obstruction to vessels resulting in congestion in some areas and anemia in others (MacCallum,[57]). The histopathological appearance is characterized by an absence of leucocytic infiltration and inflammatory reaction and the presence of a colliquative necrosis and disintegration of the liver cells. Whereas in most instances the abscesses increase in size, MacCallum[57] states that there may be attempts at healing in which there is the formation of a thick wall of granulation. The amebae die out and the majority of the fluid is absorbed, following which the abscess wall contracts down around a mortar-like material and ultimately may be replaced by scar.

SYMPTOMATOLOGY OF AMEBIC HEPATIC ABSCESS

The symptoms and signs in amebic hepatic abscess may be divided into two groups, systemic and local. Of the systemic manifestations fever is by far the most frequently encountered. Less frequently there occur chills, profuse sweating, weakness, anorexia, loss of weight, nausea, vomiting, and malaise. Chen et al[37] stated

that their patients usually had no fever when admitted to the hospital, but the average temperature in all their patients was 37.3 degrees C. In our own series of 73 cases there were 51 in which fever was present, an incidence of 69.8 per cent (Fig. 3). There were 114 cases reported in the literature[31,34,42,43,54] in which a statement was made concerning the presence of fever. To this we have added our 73, making a total of 187. Of this group, fever was present in 151 (80.7 per cent) (Table VII). Rogers[58] has emphasized that in the patients with acute abscess the fever may be of the intermittent or remittent type, whereas in the chronic cases the temperatures may be normal or even subnormal. In Manson-Bahr's[43] series of 45 cases intermittent fever was present in 35 (77 per cent), the remittent typhoid-like fever in 4 (8 per cent). From the literature[25,31,43,54] together with our own we have 174 cases in which a statement concerning chills was made. Of this group there were 43 (24.7 per cent) with chills (Table VIII). In our own series chills were present in only 10 (13.6 per cent) (Fig. 3). Loss of weight was observed in 14 of Talbot's[54] 15 cases and in our own series it occurred in 33 cases (45.2 per cent). Nausea and vomiting occurred in 14 of Young's 15 cases, whereas in our own series it occurred in only 10 cases (13.6 per cent) (Fig. 3). Weakness is frequently a prominent symptom as it was in our cases, being present in 41 cases (56.2 per cent). In spite

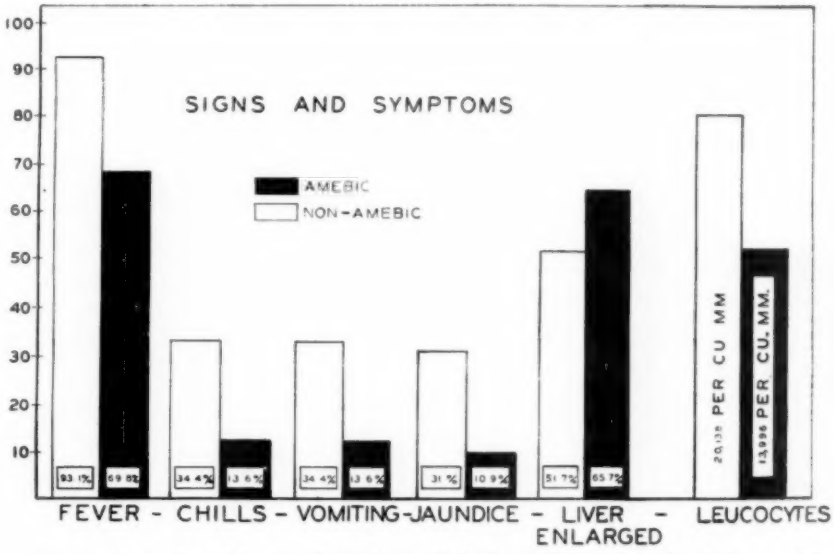

Fig. 3. Graphic representation of incidence of various clinical manifestations both in amebic and non-amebic abscesses of liver.

Table VII
INCIDENCE OF FEVER

Author	No. of Cases	Fever Present
Manson-Bahr[43]	45	39
Young[31]	15	15
Talbot[54]	15	15
Giggam et al.[42]	25	18
Boland[34]	14	13
Authors	73	51
Total	187	151 (80.7%)

Table VIII
INCIDENCE OF CHILLS

Author	No. of Cases	Chills Present
Young[31]	15	11
Talbot[54]	15	6
Murray[25]	26	14
Manson-Bahr[43]	45	2
Authors	73	10
Total	174	43 (24.7%)

of the fact that everyone is agreed that amebic hepatitis is the result of amebic infection of the colon, frequently a history of a previous diarrhea is not obtained. In a series of 318 cases collected from the literature[21,31,33,37,42,43,54,59] including our own there were 187 (58.8 per cent) cases with a positive history of antecedent diarrhea (Table IX). In our series a positive

TABLE IX
INCIDENCE OF HISTORY OF DIARRHEA

Author	No. of Cases	Diarrhea Present
Young[31]	15	7
Talbot[54]	15	6
Ludlow[59]	10	9
Murray[25]	26	14
Giggam et al.[42]	25	13
Chen et al.[37]	48	34
Thurston[33]	64	44
Manson-Bahr[43]	45	27
Tao[27]	18	2
Authors	52	31
Total	318	187 (58.8%)

history of previous diarrhea was obtained in 31 of 52 cases (59.6 per cent), although diarrhea was a symptom at the time of admission to the hospital in only 10 of the cases. The period of time between the active amebic infection of the bowel, as evidenced by diarrhea, may be extremely long, although one cannot say definitely that in the interim there is not a latent infection of the bowel, Mallory[60] and Low[61] report cases with thirty and twenty years, respectively, between the dysenteric manifestations and the development of the hepatic abscess. In one of our cases the period elapsing between the dysentery and the appearance of the liver abscess symptoms was eight years. The relatively large number of patients with amebic hepatitis who give no history of previous diarrhea may be accounted for on the basis of a slight amebic infection of the bowel and that limited to the right half of the colon, which would produce no diarrhea. Rogers[47] found in 13 of 36 cases amebic ulcers were limited to the cecum, and in 4 more extended only as far as the ascending colon. In nearly half the cases only the proximal fourth of the large bowel was involved. In 6 additional cases scars were found in the cecum and ascending colon. Thus, in 23 of 36 cases, almost two-thirds, the lesions were limited to the cecum and the ascending colon, indicating that during life amebic infection was latent. As Rogers[48] has emphasized, it is this type of amebic infection of the bowel which is associated with the solitary large abscess of the liver and not associated with the small multiple abscesses, because, as discussed under pathology, in the sloughing infiltrating type associated with diarrhea, frequently multiple abscesses occur because of the invasion of the liver by large numbers of amebae.

LOCAL MANIFESTATIONS

Pain in the upper right abdominal quadrant is probably the most constant symptom in amebic hepatic abscess. It was present in all of Young's[31] cases, in 14 of Talbot's[54] 15 cases, in 22 of Murray's[25] 26 cases, in 23 of 25 cases of Giggam et al.,[42] and in 75 per cent of Manson-Bahr's[43] series. Frequently the pain is referred to the supraclavicular region and is undoubtedly the result of involvement of the diaphragmatic pleura. This is easily understood when one realizes that the majority of amebic abscesses are located on the convex surface of the liver in contact with the diaphragm. According to Manson-Bahr[43] this is the most striking and characteristic pain, being present in the right acromial region in 20 cases, and in 2 patients it radiated to the left, in both of whom an abscess in the left lobe was present. Shoulder pain was present in one-sixth of Manson's[62] series. In our own series pain was the most important symptom, being present in 58 cases (79.4 per cent). Associated with the pain is usually pain induced by pressure, i.e., tenderness. In our own series it was present in 58 cases (79.4 per cent). Ludlow[30] described a sign

which he believes is of diagnostic importance. This sign consists of a deep-seated pain produced by a sudden thrust of the end of the finger on the liver. The most common point at which this can be elicited is about 2 inches below the right costal margin. Enlargement of the liver occurs in the majority of instances. Young[31] found it in almost all of his cases, Talbot[54] in his entire group, Manson-Bahr[43] in 75 per cent of his cases. Murray[25] noticed a swelling in the right side or in the epigastrium in 14 of his 26 cases. In our own series there was englargement of the liver in 48 (65.7 per cent) (Fig. 3). Occasionally in those instances in which a distinct bulging can be palpated there is redness and edema of the skin, which indicates involvement of the abdominal parieties. According to Stevenson[63] this occurs in about 5 per cent of cases. Jaundice occurs infrequently. It was slight in 5 of Young's[31] 15 cases, in 1 of Talbot's[54] 15 cases, and present in 3 to 4 per cent of Ludlow's[30] 100 cases. In our own series jaundice was present in 8, an incidence of 10.9 per cent (Fig. 3). Rolleston[32] states that jaundice is rarely deep when present, but usually consists merely of a tinging of the sclera. The icterus index in our cases with jaundice varied from the highest figure of 200 to the lowest of 5, with an average of 42.8. The duration of the symptoms in amebic hepatic abscess varies considerably. In Thurston's[33] cases it varied from five days to eight months, whereas in Murray's[25] series the average duration was four and a half months. In our own series the duration was as follows: The shortest was one day, the longest three years, with an average of 63.1 days. In an attempt to determine the relative number of acute and chronic abscesses we considered those patients with symptoms up to three weeks as having a chronic abscess, considering three weeks merely as an arbitrary dividing point. In the former group there were 28 cases (38.3 per cent), whereas in the latter group there were 45 cases (61.6 per cent). In 6 cases of our series (8.2 per cent) the patient had been previously treated with emetine for amebiasis.

LABORATORY FINDINGS

Rogers[2,47,48,58] and Manson-Bahr and Willoughby[64] have emphasized that in amebic hepatic abscess there is a moderate increase in leucocytes as contrasted with the marked leucocytosis which is seen in bacterial hepatitis and abscess. Characteristically, there is not a disproportionate increase in the polymorphonuclear leucocytes as is found in the pyogenic type. Manson-Bahr and Willoughby[64] found in their group of cases that the highest leucocyte count was 25,000, the lowest

TABLE X
BLOOD PICTURE

	Acute Cases	Chronic Cases	Total
Leucocyte Count			
Lowest	7,500	4,250	4,250
Highest	31,500	22,500	31,500
Average	15,460	13,000	14,000
Neutrophiles			
Lowest	70%	59%	59%
Highest	95%	90%	95%
Average	80%	75%	79%
Hemoglobin			
Lowest	50%	35%	35%
Highest	80%	82%	82%
Average	69.6%	60%	64%
Erythrocytes			
Lowest	2,650,000	1,950,000	1,950,000
Highest	4,975,000	4,590,000	4,975,000
Average	3,948,000	3,473,000	3,700,000

7500, the average 15,000 with no eosinophilia. Talbot[54] found an average of 18,000 in 14 patients. In contrast to Rogers[47] and Manson-Bahr and Willoughby,[64] he found a polymorphonuclear leucocytosis. Young[31] found the variation to be between 9000 and 30,000, with an

average of 16,000, and Candler[53] reports variations between 4700 and 28,000, with 70 to 80 per cent polymorphonuclears. Rogers[65] believes a high leucocyte count is indicative of multiple abscesses and offers a bad prognosis. Manson-Bahr[64] found that the cases with the lowest counts had the largest abscess cavities. In our own series the blood counts varied according to chronicity of the lesion, as is shown in Table x. The average for the entire series was as follows: W.B.C. 14,000, polymorphonuclear leucocytes 79 per cent, Hb. 64 per cent, and R.B.C., 3,700,000.

Amebae were found in the stool in 34.1 per cent of the series of cases of Chen et al.[37] Manson-Behr[43] observed only 2 cases in his series of 45 in which vegetative amebae were present in the stools, as contrasted with Tao's[27] 77.7 per cent positive finding in his series. In our own series statements concerning the presence or absence of amebae in the stool were made in 36, of which 13 were positive (36.1 per cent), and 23 (63.8 per cent) negative (Fig. 2).

X-RAY DIAGNOSIS

One of the most important and reliable aids in the diagnosis of liver abscess is roentgenography. We have repeatedly corroborated the findings of Granger[66] and feel that the characteristic roentgenologic changes are of inestimable value. Granger[66] emphasizes the importance of the characteristic changes in the contour of the diaphragm in liver abscess alone and in that associated with subdiaphragmatic suppuration. He states that in uncomplicated liver abscess there is distinct bulging of the diaphragm and pointing upward into the lower lung field, which is almost pathognomonic of liver abscess, although a gumma of the liver may produce the same picture (Fig. 4). He is able to distinguish the subphrenic abscess complicating a liver abscess from those due to other causes. In the former there is an obliteration of the cardiophrenic angle in the antero-posterior roentgenogram and obliteration of the anterior costophrenic angle in the lateral view (Fig. 5), whereas in the latter there is an obliteration of the costophrenic angle in the antero-posterior view and obliteration of the posterior costophrenic angle in the lateral roentgenogram (Fig. 6). Pancoast,[67] Dickinson,[68] and Love[69] emphasize the importance of elevation and immobility of the diaphragm in amebic abscess of the liver. These authors have stressed the importance of fluoroscopy and the examination of the patient in the upright position when such is possible. We agree with Pancoast[67] that the lung reaction is of diagnostic importance when present and usually indicates an amebic infection, because these abscesses occur most frequently on the convex surface of the liver in close contact with the diaphragm. The value of *x*-ray in the diagnosis of amebic hepatic abscess is well exemplified in our own series. In 57 cases in which *x*-rays were made, the *x*-ray diagnosis was positive in 50, an incidence of 87.7 per cent. Wilmoth[70] advocates aspiration of the pus from hepatic abscesses and the introduction of an iodized oil into the cavity in order that the size, shape and position of the abscess may be visualized on the roentgenographic plate. Reeves and Appel[71] and Yater and Otell[72] suggest and report the use of thorium dioxide in amebic abscess of the liver in which the abscess appeared on the plate as a negative shadow. Because of the insufficient knowledge of the end-effects of thorium dioxide administration, it hardly seems justifiable at the present time to use such a drug in the diagnosis of a condition which can be so readily diagnosed. That this statement is not incorrect is borne out by the percentage of cases in which a correct roentgenographic diagnosis was made in our series. This opinion is also shared by Rothenberg and Linder.[73]

DIAGNOSIS

The diagnosis of amebic hepatitis and abscess of the liver is not difficult if the condition is kept in mind. In a patient

with persistent enlargement of the liver associated with pyrexia one should always consider the possibility of amebic infection. If in addition to these findings, there is slight leucocytosis without concomitant proportionate increase in polymorphonuclear leucocytes, one should be even more suspicious of an amebic infection, as suggested by Rogers.[2] A history of previous dysentery is confirmatory evidence, but the absence of such a history in no way eliminates the possibility of amebiasis. The presence of pain and tenderness in the right upper abdominal quadrant, especially limited to the liver or radiating to the right shoulder is corroborative. Such a clinical picture together with the characteristic fluoroscopic and roentgenologic findings already described will justify a diagnosis of liver abscess. If, in addition, one finds on careful examination of the stools either active amebae or encysted forms, a positive diagnosis can be made. Here again a negative finding does not rule out amebiasis as a cause of the liver involvement. Exploratory aspiration of the liver for the detection

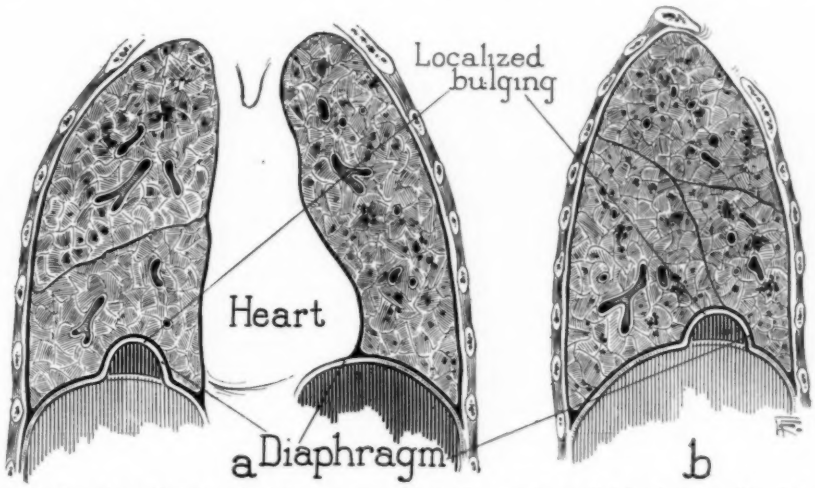

Fig. 4. Diagrammatic drawing showing bulging upward into lower lung field of a localized hepatic abscess. In anterior-posterior roentgenogram (a) bulging is in medial portion of lung field, whereas in lateral roentgenogram (b) it is located more anteriorly than posteriorly.

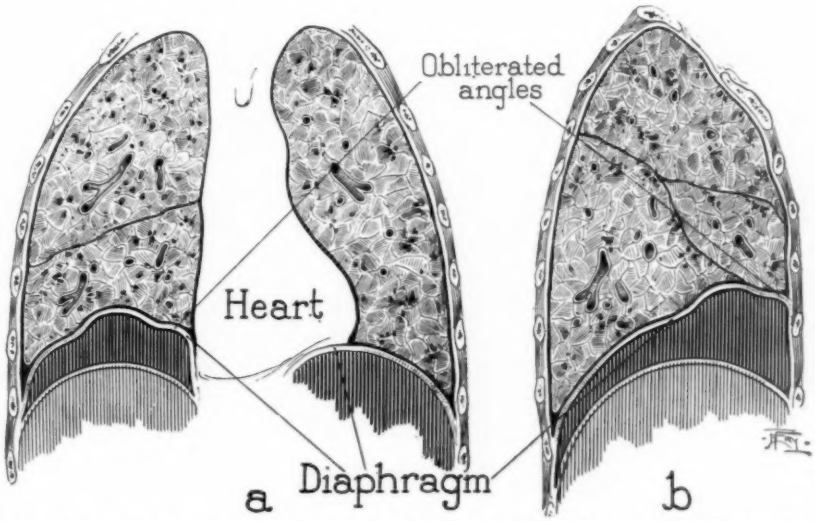

Fig. 5. Diagrammatic drawing showing characteristic elevation of diaphragm in subphrenic abscess following rupture of an hepatic abscess. In anterior-posterior roentgenogram (a) there is obliteration of cardiophrenic angle, costophrenic angle being uninvolved. In lateral roentgenogram (b) there is an obliteration of anterior costophrenic angle, posterior costophrenic angle being free.

of pus is important in the diagnosis. If it could be determined with certainty that the liver lesion is an amebic abscess not are sterile. We cannot agree with Young[31] that the danger of infecting the pleural or peritoneal cavity has been overemphasized,

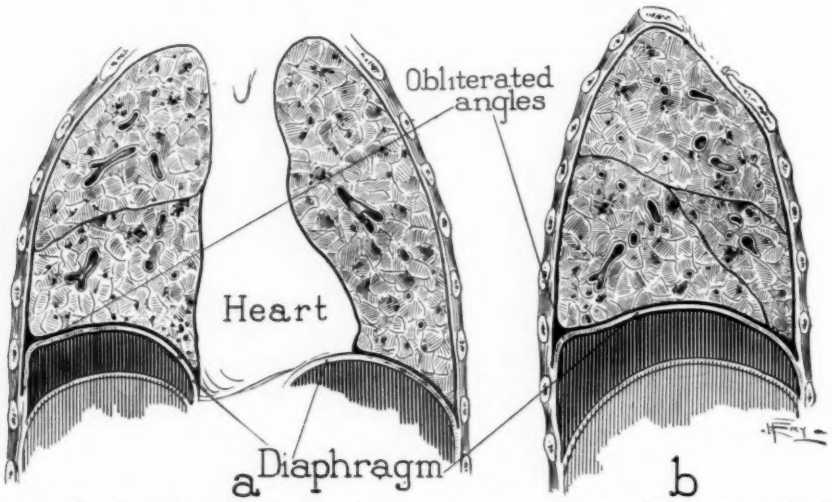

FIG. 6. Diagrammatic drawing showing changes in roentgenogram obtained in subphrenic abscesses resulting from intra-abdominal infection aside from liver abscess. In anterior-posterior roentgenogram (a) there is obliteration of costophrenic angle, whereas in lateral roentgenogram (b) there is an obliteration of posterior costophrenic angle.

secondarily infected, the site of aspiration would be inconsequential. Because it is not possible to determine whether a liver abscess is not a pyogenic one or, if originally amebic, that it is not secondarily infected, one should always assume that the abscess cavity contains pyogenic bacteria, and the aspiration done in such a way that an uninvolved portion of the peritoneum or the pleural cavity is not traversed by the aspirating needle. It is also imperative that aspiration is done in the operating room so that if pyogenic organisms are found in the aspirated material, an immediate open drainage can be performed. In order to determine the presence or absence of pyogenic organisms, a smear should be made immediately after aspiration and if a large quantity of such organisms are found, open drainage should be instituted. In those abscesses located posteriorly it is important that the needle be introduced below the costophrenic angle and inserted upward into the abscess cavity. Under no circumstances should the pleural cavity be traversed even though many are of the opinion that this precaution is not justified, because uncomplicated amebic abscesses

sized, and are inclined to believe that Young is fortunate in being able to base his opinion upon observations made in uncomplicated amebic abscess. The aspiration of the characteristic chocolate-sauce pus is of diagnostic importance. According to Constantini,[56] Chen et al.,[37] and Manson-Bahr,[43] such a finding is pathognomonic of amebic abscess of the liver. Ludlow[30] stresses the fact that in aspirating amebic abscess of the liver the needle should not be introduced more than 3 to 4 inches because of the danger of injuring a large vessel. It is at times necessary to attempt multiple punctures before pus is encountered. If multiple aspirations are done, it is important to remove the needle entirely before reintroducing it rather than changing the direction of the needle at the original site. In this way extensive injury to the liver will be obviated. Either local analgesia or general anesthesia may be used. In our own series of 73 cases, a correct preoperative diagnosis was made in 50, an incidence of 68.4 per cent. The value of *x*-ray diagnosis is well illustrated in our series, because the only cases in which a correct preoperative diagnosis was made

were those in which roentgenograms were obtained and the positive diagnosis made on the roentgenogram. In many of these cases the clinical picture was such, however, that the diagnosis could be made without the use of the *x*-ray.

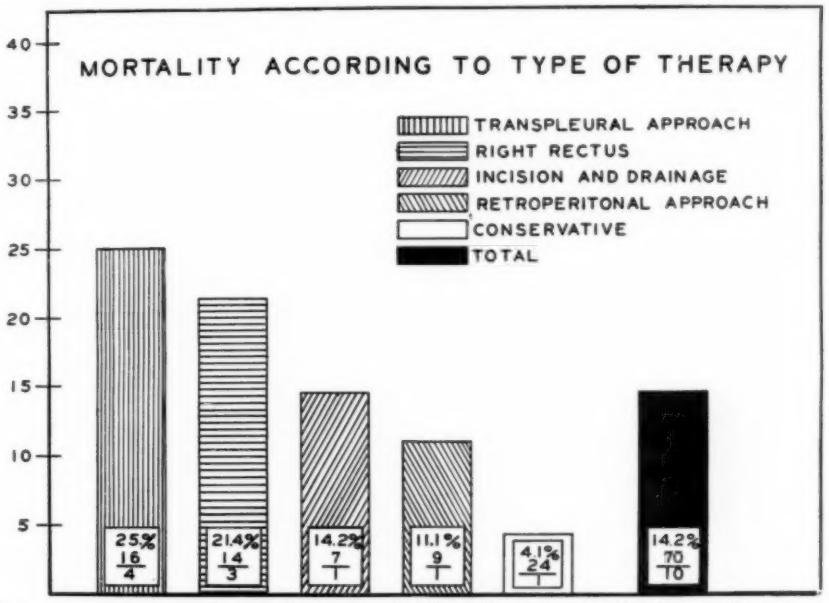

Fig. 7. Graphic representation of mortality rate obtained in present series by various types of therapy.

PROGNOSIS

Prognosis in amebic abscess of the liver depends upon a number of factors. These are: (1) the multiplicity of lesions in the liver, which in turn are dependent upon the severity of the amebic infection; (2) general resistance of the patient; (3) secondary infection of the abscess cavity; and (4) the type of treatment employed. According to Craig,[74] the prognosis in amebic liver abscess is grave. He believes that the mortality rate in these cases is at least 25 to 30 per cent, if in the statistics multiple abscesses are included. The importance of closed drainage in uncomplicated amebic abscess of the liver is emphasized by the results obtained by those who employ repeated aspiration. Rogers[75] showed that the mortality rate of 56.8 per cent which was obtained in cases with open drainage was decreased to 14 per cent in those cases in which closed drainage was used. The greater mortality rate in those cases treated by open drainage is due undoubtedly to the secondary infection of the abscess cavity from which there is considerable absorption and extension of the process into contiguous portions of the liver. The process is not unlike the secondary infection which occurs in tuberculous abscesses which are opened. Only by keeping the abscess cavity closed is there any possibility of maintaining a sterility of its contents. The value of closed drainage is shown in Chatterji's[76] series in which there was a mortality rate of

Table XI
MORTALITY IN OPEN OPERATION

Author	No. of Cases	Died
Rogers[75]	2661	1511
Futcher[28]	27	19
Solonof[78]	1094	328
Talbot[54]	4	1
Thurston[33]	6	0
Ludlow[30]	100	10
Perard[79]	24	3
Young[31]	15	1
Boland[34]	13	5
Chen et al.[37]	25	8
Love[69]	20	13
Authors	46	9
Total	4035	1908 (47.2%)

1.6 per cent. It is probable, however, that Chatterji's cases were carefully selected and did not include those with multiple

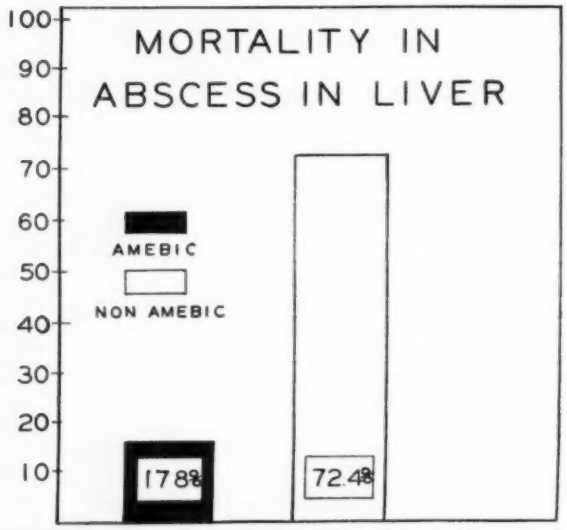

FIG. 8. Graphic representation of total mortality rate in amebic and non-amebic liver abscesses.

abscess, because he[77] advocated open drainage and not closed aspiration in cases with 2 or more abscesses. The mortality rate in amebic liver abscess has been definitely lowered as a result of early diagnosis, the preoperative and postoperative use of emetine, and the substitution of aspiration for open drainage. In our own series a mortality rate of 19.5 per cent was obtained in 46 cases in which an open operation was done. In a series of 24 in which conservative treatment was used there was one death, a mortality rate of 4.1 per cent (Fig. 7). In a series of 4035 cases collected from the literature* including our own in which open operation was done, 1908 (47.2 per cent) (Table XI) ended fatally, whereas in a series of 459† in which conservative treatment was used only 32 (6.9 per cent) died (Table XII). There were 13 deaths in our series of 73 cases resulting in a total mortality of 17.8 per cent (Fig. 8).

TABLE XII
MORTALITY IN CLOSED OPERATION

Author	No. of Cases	Died
Rogers[75]	111	16
Chatterji[76]	186	3
Talbot[54]	11	0
Manson-Bahr et al.[80]	15	0
Thurston[33]	58	9
Ludlow[59]	10	0
Perard[79]	22	1
Chen et al.[37]	22	2
Authors	24	1
Total	459	32 (6.9%)

TREATMENT

The reduction in the mortality rate in amebic abscess of the liver has been due to two factors: (1) the introduction of the specific therapy, emetine, both preoperatively and postoperatively, and (2) the appreciation that amebic abscesses of the liver are sterile and that every precaution should be made to prevent their contamination with pyogenic microorganisms. Rogers[75] found prior to 1907, during a fourteen year period before the introduction of ipecac, that in 2661 reported cases of amebic abscess there was a mortality rate of 56.8 per cent. These patients were all treated by open drainage of the abscess. As contrasted with these figures are the results obtained in a similar series of cases in which ipecac and closed drainage were used. Of 111 cases there were 16 deaths, a mortality rate of 14.4 per cent. Within two years after adoption of ipecac by the British Army in India, the admission rate for liver abscess fell from 2.4 to 1.4 per thousand troops, the death rate from 1.3 to 0.48 per thousand, and the case mortality from 54 to 34.3 per cent (Allen[81]). Older surgeons in treating amebic abscesses of the liver observed that following the open drainage of the abscess the patient did well for from one to three days, and then developed signs of toxemia, which became progressively worse and was followed by death. The importance of this was not appreciated until Rogers's[2] early investigations showed

* See ref. 28, 30, 31, 33, 34, 37, 54, 69, 75, 78, 79.
† See ref. 33, 37, 54, 59, 75, 76, 79, 80.

that the "pus" in amebic abscess of the liver was sterile in 86 per cent of instances. Rogers[75] is of the opinion that in the large, thick-walled amebic abscess destruction of the liver does not progress, although the abscess may get larger and compress the liver substances. If, however, the abscess cavity becomes secondarily infected, which invariably occurs if open drainage is instituted, the microorganisms are apt to penetrate the limiting wall and result in an extension beyond the abscess itself into the surrounding liver parenchyma. It is because of this invasion of the uninvolved portions of the liver that the patient develops a severe and frequently fatal toxemia. The infection of such a sterile abscess with its marked deleterious effects has been compared by Rogers[75] and Neligan[82] to the change which occurs in a tuberculous abscess that has been drained and in which secondary infection occurs. Whereas the majority of amebic abscess of the liver are surgically sterile, there may be some microorganisms in the pus, which, however, are not virulent. Only exceptionally are virulent organisms obtained from the abscess at the time of aspiration or at the time of drainage. The secondary infection occurs following open drainage in spite of meticulous care being used to prevent contamination. The advantage of aspiration of the abscess combined with emetine therapy has been emphasized by many investigators (Manson-Bahr et al.[80]; Thurston[33]; Ludlow[59]). We have been able to collect from the literature, including 46 cases of our own, 4035 cases in which open operation has been performed. In this group there were 1908 deaths, a mortality rate of 47.2 per cent (Table XI). We have also collected from the literature, including 24 cases of our own, 459 cases treated by conservative measures; viz., closed drainage and the administration of amebicides. There were 32 deaths, a mortality rate of 6.9 per cent (Table XII). These figures illustrate graphically the advantage of the conservative treatment over the open drainage of amebic liver abscesses. In our own series there were 46 patients in which an open operation was done with 9 fatalities, a mortality rate of 19.5 per cent, which indeed compares favorably with the mortality rate obtained from the collected cases in which open operation was done; viz., 47.2 per cent. In our own series there were 24 cases in which conservative treatment was used with 1 death, a mortality of 4.1 per cent. The 1 death in the group of cases treated conservatively was probably avoidable, because in this instance aspiration was performed on a seventy-year-old man without previous treatment with emetine, a procedure which Rogers[48,75] has repeatedly warned against, because of the danger of hemorrhage during the acute stage of hepatitis with marked congestion of the liver. Hatch[83] also calls attention to this danger and reports a case in which fatal hemorrhage resulted from such aspiration. In our case, already mentioned, typical amebic chocolate-sauce material was obtained from the abscess at the time of the operation. Several hours later the patient went into shock and died the following morning. At autopsy a very large abscess of the liver together with a marked myocarditis, nephritis, edema, and congestion of the lungs was found. It is our opinion that had preliminary emetine therapy been used this tragedy might have been averted. In our series of 73 cases of amebic abscess there were only 3 which were not treated. These 3 patients were moribund at the time of admission to the hospital and died before therapy could be instituted. Of the 70 receiving treatment, there were 24 treated conservatively by emetine administration and aspiration (Fig. 7) with 1 death, a mortality rate of 4.1 per cent. This fatality has already been described. Of 46 cases in which open drainage was used, the retroperitoneal approach was employed in 9. In this group there was 1 death, a mortality rate of 11.1 per cent (Fig. 7). This technique has been described in detail in previous reports.[84,85] In the fatal case

there were 3 distinct abscesses of the liver with considerable secondary infection. As all abscesses were not drained at the first operation, a second operation was required and following this the patient developed pneumonia and died. There were 14 patients in whom a right rectus incision was used for the drainage of the abscess. Three of these patients died, giving a mortality rate of 21.4 per cent (Fig. 7). One of these patients died of spinal analgesia on the operating table, and, therefore, must be considered as an operative death. Another patient had a generalized peritonitis from perforation of an amebic ulcer in the descending colon. This patient also died while being operated upon. The third patient, who was very weak at the time of admission, had multiple abscesses associated with extensive ulceration of the large bowel. Two operations were required because of incomplete drainage. At autopsy an infection of the subphrenic space and peritonitis were found as well as a lobular pneumonia. In our series there were 16 cases in which a transpleural drainage by means of rib resection was done. In this group there were 4 deaths, a mortality rate of 25 per cent (Fig. 7). In one of these fatal cases the patient had a two-stage drainage of the abscess and died approximately three weeks after the second operation. At autopsy two abscess cavities in the right lobe of the liver, an extensive pneumonitis on the right side, pericarditis, and nephritis were found. The second fatality in this group occurred in a man who was admitted to the hospital with a bronchohepatic fistula. He was treated conservatively for a short period of time, left the hospital, and returned two months later, at which time a transpleural drainage was performed. This was undoubtedly an error in judgment, because in such an instance it is generally agreed that a continuation of conservative therapy is to be preferred. Immediately following the operation the patient went into a semicomatose state and never recovered, dying two days postoperatively. In the third fatal case in which a rib resection was performed the operator was unable to find the abscess. At autopsy seventeen days postoperatively a large abscess in the dome of the right lobe of the liver was found which had perforated into the right pleural cavity with the production of an empyema, collapsing the right lung. Undoubtedly the error was made in this case in not administering emetine preoperatively and postoperatively, as the patient did not receive emetine at any time. The fourth fatality in this group was a patient who was admitted to the hospital following an injury and was treated for an apparent contusion of the thorax and upper abdomen. He was discharged from the hospital two weeks later with still some pyrexia. Two weeks later he was readmitted, at which time a transpleural drainage was performed and an amebic abscess of the liver drained. He died eight days postoperatively from a peritonitis resulting from a rupture of an amebic ulcer of the colon. Autopsy showed pleural effusion and a serous effusion of the pericardium. Rogers[48] in 172 cases found that the mortality rate in those cases in which drainage was instituted through the pleural cavity was 73 per cent; in the right lobe abscesses in which the drainage was instituted just beneath the right costal margin the mortality rate was 59 per cent, whereas in small epigastric left lobe abscesses the mortality rate was 12 per cent. In our series of cases there were 7 in which simple incision and drainage of a pointing abscess was made. There was 1 death, a mortality rate of 14.2 per cent (Fig. 7). This fatality occurred following incision and drainage in a patient whose abscess was drained through a kidney incision. A large quantity of pus was evacuated. The patient, however, died nine days after the operation. Autopsy showed a large abscess of the liver.

The low mortality rates obtained by the simple incision and drainage and the retroperitoneal approach, 14.2 per cent and

11.1 per cent, respectively, as compared with the high mortality rates following the transpleural and transperitoneal approaches; viz., 25 per cent and 21.4 per cent, respectively, illustrate the advantage of avoiding contamination of a serous cavity even when employing drainage in an amebic abscess and do not support Young's[31] contention that "the danger of infecting the pleural or peritoneal cavity has been overemphasized." However, the comparatively low mortality rates which have been obtained by the extrapleural and extraperitoneal drainage cannot be compared with the results obtained by the conservative treatment. Undoubtedly, many, if not all, of the patients in whom an extraperitoneal drainage was done died of secondary infection of the amebic abscess. However, in one of these cases the abscess was obviously infected at the time of operation precluding the use of conservative therapy. It is our opinion that every case of suspected amebic abscess should be given the advantage of a course of emetine therapy before any other procedure is used unless there is apparently danger of rupture of the abscess. We employ emetine, grains 1 (gm. 0.065), daily two to four days before aspiration. Frequently in small abscesses no other therapy will be necessary. In the majority of instances, however, emetine will not suffice and some measure should be used to evacuate the abscess contents. Rogers,[48] Hodson,[86] and Hartman-Keppel[87] have emphasized that even large liver abscesses may completely disappear under emetine therapy alone. We, however, agree with Rogers[48] that the pus should be evacuated in some way in order that it is not necessary for the body to remove it by absorption, which would only increase the time of convalescence. After the preliminary emetine treatment, aspiration of the liver should be done preferably under local analgesia. Occasionally, however, if difficulty is encountered in locating the abscess, a general anesthetic might be used, but it is seldom necessary.

The technique of aspiration depends considerably upon the clinical manifestations. In those instances in which there are localizing signs and pointing of the abscess, the aspirating needle should be introduced directly over the mass. Otherwise, if no localizing signs are present, frequently multiple aspirations should be made. Here, too, it is imperative that the needle be introduced in such a way that the pleural and peritoneal cavities are not traversed. This can be accomplished by introducing the needle in the tenth intracostal space in the anterior axillary line and directing it upward, medially, and backward. As emphasized by Manson-Bahr et al.[80] and Ludlow[59] the needle should be introduced not more than 3½ to 4 inches, because of the danger of injuring the large portal radicals. Occasionally an abscess can be entered by introducing the needle below the twelfth rib and extending it upward and anteriorly. After pus is obtained by means of the aspirating needle, it is frequently necessary to introduce a trocar, because difficulty may be encountered in emptying the abscess cavity on account of the extreme thickness of the contents. Kilner[91] emphasizes that frequently the thick contents of the abscess will cause blockage of even a trocar, giving a false impression that the abscess has been evacuated. When the abscess cavity is found as much of the "pus" should be evacuated as possible. It is neither necessary nor desirable to introduce any substance into the abscess cavity as it has been shown by Rogers,[58] Talbot,[54] and Chatterji[77] that the use of irrigating solutions and amebicides is valueless. An immediate smear and culture of the pus should be made. If a large number of pyogenic organisms are found in the smear and if a positive culture is obtained in the majority of instances an open drainage of the abscess is necessary, because in such an instance the secondary infection of the amebic abscess is of more importance than the original abscess itself. When such a procedure becomes necessary, it is im-

portant that the emetine therapy be continued and that the abscess be drained in such a way that there is no contamination of either the pleural or peritoneal cavities. This is best accomplished by the retroperitoneal operation.[84] In those cases in which the abscess is found to be sterile, open operation is contraindicated. After the aspiration of the pus from the abscess an intramuscular injection of emetine should be given, because following the release of the tension within the abscess according to Rogers[75] and Talbot[54] there occurs an exudation into the abscess of lymph containing the injected emetine. Emetine should be administered in grain doses daily until 6 to 10 grains (gm. 0.39 to 0.65) have been given. According to Leake[88] the amount of emetine administered over any given period of time should not exceed 10 mg. (1/6 gr.) per kilogram of body weight. A maximum dose for a person weighing 150 pounds would be approximately 10 grains. Emetine should be used cautiously because as shown by Rinehart and Anderson,[89] working in Leake's laboratory, it produces in the experimental animal severe injury to the cardiac muscle. The lesion found in the heart is quite similar to the Aschoff reaction of acute rheumatic fever. Leake[88,90] is of the opinion that although other amebicides, acetarsone, carbarsone, treparsol, chinofon, and vioform, are safer and more efficient in the treatment of intestinal amebiasis, they should not be used in amebic hepatitis and liver abscess, as they themselves are toxic to the liver.

COMPLICATIONS

The complications of amebic abscess of the liver are the result of rupture or direct extension into one of the adjacent viscera, secondary inflammation and suppuration, or thrombosis and embolism. The complications are frequently indicative of negligence on the part of the patient, but not uncommonly signify unproficiency and inadvertence of the physician.

The most frequent complications are pleural and pulmonary involvement. Only a succinct consideration of these complications will be accounted here, as a more detailed study of this subject will follow in a subsequent report.[91a] The estimations given by different writers vary from 3 per cent, as reported by Strong,[92] who found that in 100 fatal cases, one involved the lung and 2 the pleura, to 40.8 per cent, 11 of a series of 27 cases reported by Thompson.[93] Rather high percentages, 33.3 and 20, respectively, of lung complications were also reported by Futcher[28] and Harrington.[94] As contrasted to this, Cyr,[95] Talbot,[54] and Manson-Bahr[43] found pulmonary involvement in 10.4 per cent, 6.6 per cent, and 11 per cent, respectively. In our own series the percentage of lung involvement more closely resembles the latter reports, as there were 7 (9.5 per cent) of 73 amebic liver abscesses which ruptured into the lung and one (1.3 per cent) that ruptured into the pleura. This is in marked contrast to the estimate given by Vergoz and Hermanjat-Gerin,[96] who report 28 (35 per cent) ruptures into the pleura in a series of 80. Murray[25] also found a rather high incidence, 15.3 per cent. On the other hand, rather low incidences (9 per cent, 7.4 per cent, and 5.5 per cent) are recorded by Ludlow,[30] Futcher,[28] and Cyr,[95] respectively. The explanation of the relative frequency of these complications is attended with little difficulty. There are two possible routes of secondary extension: (1) usually by direct extension through the diaphragm, and (2) rarely by embolism. The latter route of extension is very infrequent and merits only passing mention. Brown[97] reported one case that undoubtedly occurred in this manner. As discussed under morbid anatomy, the majority of amebic abscesses of the liver occur on the convex and posterior portion of the right lobe of the liver. As the abscess enlarges, its periphery approaches the diaphragm, gradually incorporating it in the wall of the abscess. If an accompanying pleurisy with adhesions has obliterated the pleural cavity, the abscess ruptures into

the base of the right lung. If the process is more rapid and no antecedent pleurisy has developed, the abscess will rupture into the pleural cavity. After rupturing into the lung, the abscess frequently communicates with a large bronchus and drainage through a bronchohepatic fistula results in copious expectoration of chocolate-colored sputum in which careful search will frequently reveal the amebae.

In our series of 7 bronchohepatic fistulae, 6 were treated conservatively with recoveries in all. One patient treated conservatively, but incompletely, was discharged from the hospital and returned after two months. Conservative treatment was again instituted for only nine days, following which a transpleural drainage was performed. Immediately following the operation, the patient became comatose and died two days postoperative. Although our 8 cases with thoracic complications comprised too small a series on which to base conclusions the results are very suggestive of the value of conservative therapy in amebic bronchohepatic fistulae. The mortality rate in our 7 cases of bronchohepatic fistulae was 14.2 per cent, which is low as compared with other statistics. The case with rupture into the pleural cavity ended fatally. This patient had been operated upon, but at operation the liver abscess was not found. Rupture into the pleural cavity subsequently occurred, and at autopsy a large abscess of the liver with perforation into the pleurae was present.

Rupture of an amebic hepatic abscess into the peritoneal cavity occurred in 39 of Cyr's[95] 159 cases of ruptured amebic abscess in a total series of 563 cases of hepatic abscess. Thierfelder[98] observed this complication in 23 of his 170 collected cases in which rupture had occurred. This incidence is too high, because at the time these observations were made (1880), undoubtedly many cases of amebic abscess were not recognized and were neglected, permitting extension and perforation before therapy was instituted. Ludlow[30] observed this complication in 5 of his 100 cases, an incidence of 5 per cent. As in the pleural cavity, frequently adhesions are formed on the surface of the liver so that perforation into a free peritoneal cavity does not occur. In this way a walling-off of the abscess prevents the free perforation. Following exertion, however, there may be rupture into the virgin peritoneal cavity, as in the case reported by Biggam and Rajab.[99] In such an instance, due to the sudden flooding of the peritoneal cavity with the amebic pus, absorption of toxic material may occur, producing a picture of shock. As the toxemia is not as marked as that seen in cases of pyogenic infection, the prognosis is much better. Because of this, Biggam and Rajab[99] advocate aspiration of the peritoneal fluid and the institution of specific therapy, which was successful in their case. According to Waring,[44] most of these cases are fatal. In our series there were 6 patients in whom a rupture into the peritoneal cavity occurred with 2 fatalities, a mortality rate of 33.3 per cent. In 2 of these there was localized peritonitis, resulting from walling-off of the process before rupture into the peritoneal cavity occurred. Both of these patients were operated upon and one died. The one who recovered had a subphrenic abscess, which was operated upon successfully by the retroperitoneal approach. There were 4 patients in whom the perforation occurred into the virgin peritoneum, resulting in a generalized peritonitis, and one of these recovered. It is noteworthy, however, that the 3 patients who did not recover were not operated upon, whereas the one that did recover was operated upon. From this it is suggestive that rupture of an amebic abscess, especially into a free peritoneal cavity, is an indication for immediate operation with evacuation of the contents This contention is at variance with Biggam and Rajab,[99] who believed that conservative treatment, which consists of aspiration of the peritoneal fluid and specific therapy, should be used. Rupture into the intestinal tract occurs infrequently, and in our

series this complication was not present in any of the cases. Rupture through the abdominal wall is a rare complication, such cases, however, having been observed by Chang Chi Ch'eng[100] and Ludlow.[30] Cerebral abscess, rupture into the portal vein, inferior vena cava, and hepatic veins have been reported, but are rare complications. However, Runyan and Herrick[101] observed 2 cases of cerebral abscess complicating amebic abscess of the liver in their series of 21 cases. The infrequency of this last complication is probably due to the fact that emboli, being carried through the hepatic veins, are more apt to become lodged in the pulmonary capillaries than to be carried through into the systemic circulation. Futcher[28] in the series of 27 cases of amebic abscess of the liver observed rupture into the inferior vena cava in 3 instances. This, too, may be explained on the basis of late diagnosis and probably due to lack of therapy, Futcher's[28] report being made in 1903 before the treatment of amebiasis was well understood. This contention is also corroborated by the 70 per cent mortality which Futcher[28] reports. It is our opinion that the majority of these complications should never be allowed to occur and that they represent a neglected case of amebic hepatitis and amebic abscess of the liver.

SUMMARY

1. A study of 73 cases of amebic liver abscess is presented.

2. The average age was forty-four years. The ratio of males to females was 8 to 1.

3. Fifty-nine and six-tenths per cent gave a history of an antecedent diarrhea; 17.4 per cent showed amebae in the abscess and 36.1 per cent showed amebae in the stool in those cases in which these examinations were performed.

4. The principal symptoms and signs were pain and tenderness, being found in 79.4 per cent. Fever and enlargement of the liver were found in 69.8 and 65.7 per cent, respectively. A diagnosis of amebic hepatitis was made preoperatively in 68.4 per cent. Positive *x*-ray diagnoses were made in 87.7 per cent.

5. The prognosis in amebic hepatitis depends largely upon the presence of secondary infection and the type of operation used. In a series of 4035 cases collected from the literature, and including the authors' own cases, in which open operation was used, 1908 (47.2 per cent) ended fatally. In a similar series of 459 treated conservatively there were 32 deaths, a mortality rate of 6.9 per cent.

6. In the authors' series of 70 cases in which treatment was used, 10 succumbed, 14.2 per cent mortality. Forty-six were operated upon with 9 deaths, a mortality rate of 19.5 per cent. Of these 16 were operated upon, employing the transpleural approach, with 4 deaths, a mortality rate of 25 per cent. Fourteen were operated upon through a right rectus incision with a 21.4 per cent mortality rate. Seven had simple incision and drainage over a presenting mass with a 14.2 per cent mortality rate. Nine had retroperitoneal operations with an 11.1 per cent mortality.

7. The lowest mortality rate (4.1 per cent) was obtained in 24 cases treated conservatively by aspiration and the use of amebicides.

8. The treatment of amebic hepatic abscesses which are not secondarily infected consists of aspiration and the use of amebicides and open operation should be employed only in those cases in which there is infection with pyogenic microorganisms.

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[For Remainder of References see p. 243.]

CONGENITAL NON-PARASITIC SINGLE CYST OF THE LIVER*

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CONGENITAL non-parasitic single cyst of the liver is a comparatively rare pathological condition, I having been unable to find only 61 similar cases reported in the literature. Hence, it does not seem out of place to report a case which occurred recently in my service at St. Peter's Hospital.

The case was entered on Dr. Kircher's service at this Hospital and he in turn transferred the patient to my service. The history of the case is as follows:

Mrs. Z. twenty-eight years of age, married, housewife.

Chief complaint pain in abdomen and vomiting. Since birth of first child ten years ago has had intermittent attacks of sharp knife-like pain in lower abdomen occurring about once a week, for first four or five years. After that, attacks became less frequent, occurring about once a month. No vomiting with attack. No relation to meals. Constipated all her life. No radiation of pain to back or sides. No loss of weight. No blood in stools. No belching. No jaundice. The week before admission to hospital patient was seized with a severe attack of pain in abdomen accompanied by chills and vomiting. Had previously noted what she called a lump in the right side of her abdomen which seemed to settle back in place when she lay down. At time of last attack she said the right side of her abdomen seemed higher than the left. Her physician sent her to the hospital for operative intervention.

Family History. Father dead, cause unknown. Mother living and well. Three brothers living and well. Four sisters living and well. No tuberculosis, cancer, diabetes or kidney trouble in family. One child died at fifteen months of convulsions. One sister has heart trouble, nature unknown.

Personal History. Childhood: scarlet fever at eight or nine years. No other illnesses. Adult history: negative for surgery and medicine. Married eleven years. Husband living and well. One pregnancy, one child ten years old. Normal delivery. Menstrual periods have always been irregular. No pain or discomfort. Has had leucorrheal discharge for past four years. Not foul in odor.

Physical examination practically negative except for abdomen. Examination of the abdomen by inspection revealed an enlargement of the right side in the iliac region and upon palpation a tumor mass the size of a good-sized grapefruit could be felt. This tumor mass was freely movable and could be pushed to almost any place in the pelvic cavity.

Bimanual examination revealed the pelvic organs normal except for this tumor which could be pushed down into the pelvis on the right side and appeared to spring from the right ovary and tube. The kidney on right side could not be palpated, but the urine was normal, both chemically and microscopically.

The roentgen-ray examination report of abdomen and chest made by Dr. Murnane December 19, 1931, was as follows: Abdomen: Flat plate of abdomen. Shows no outline of tumor mass in the right upper or lower abdominal quadrant, however, there is a definite and marked displacement of the descending colon to the left.

Chest: Thorax is symmetrical, mid-shadow is normal, diaphragm slightly tenting of right leaf. Markings throughout lung fields are normal. Slight accentuation of the hilum shadow but changes are all fibrous in character. No hyperplasia of lymphatic structures. Other markings throughout chest are slightly accentuated, but within normal limits. Diagnostic impression: Chest is negative for pathology.

Another *x*-ray made December 21, 1931 of the abdomen was as follows: Gall bladder area negative for opaque stone. Swallowing movements are normal, esophagus normal. Stomach,

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sthenic in type, is displaced to the left and occupies a vertical position in the left upper abdominal quadrant. In the erect position the stomach assumes an almost entirely vertical position, the pylorus and duodenal bulb being at the lowermost portion. It is drawn into this position by attachment to the mass in the right lower abdominal quadrant. In the prone position tension is relaxed and stomach is allowed to resume a normal position. Jejunum is markedly crowded to the left.

Roentgenograms made fifteen minutes after fluoroscopic examination record previous findings and the barium-filled coils of jejunum and ileum are displaced to the right by a spherical mass in the right lower abdominal quadrant. In the six hour plate the half of meal is in the descending colon. Stomach is empty and with very slight retention in terminal coils of ileum. The latter are raised out of the pelvis apparently by a tumor mass in pelvis. Cecum, ascending colon, descending colon, transverse colon are normal.

Diagnostic impression: Tumor mass in the pelvis and right lower abdominal quadrant, extra-enteric.

A diagnosis of an ovarian cyst with a long pedicle was made, though the possibility of a mesenteric cyst or cystic kidney was kept in mind. A cyst of the pancreas was also thought of, but not seriously.

Operation was advised and accepted.

Operation December 26, 1931. Patient anesthetised under spinocaine. A right rectus incision was made on a level with the umbilicus extending for an inch above and 2 inches below it. Upon opening the abdominal cavity a cyst the size of a large grapefruit was found, lying apparently free in the abdominal cavity and resting on the brim of the pelvis. Further exploration of the abdominal cavity showed the pelvic organs normal and that the cyst evidently arose from the sulcus, between the right and middle lobes of the liver, near the gall bladder. There was a fairly good sized pedicle the size of one's thumb and this with the cyst was enucleated from the liver tissue with very little hemorrhage. The cyst was removed entire without rupture of its contents.

The wound was closed in the usual manner without drainage, and the patient left the hospital January 16, 1932, completely recovered and has remained well ever since.

The pathological report and discussion of Dr. Wright, former Director of the Bender Laboratory and now Professor of Pathology and Bacteriology at the Albany Medical College is as follows:

Solitary, non-parasitic cysts of the liver are rare. In 1923 Jones,[1] after carefully reviewing the medical literature up to that time, found 61 cases in which such cysts had been discovered at operation. Some of these had been diagnosed before laparotomy. Others were found and diagnosed after the abdomen was open. In addition still other cases of single, non-parasitic cysts have existed unsuspected during life and have been discovered only casually during the conduct of a postmorten examination. Such cysts, therefore, may be present during life without producing symptoms or at most producing so little in the way of symptomatology that diagnosis is never made. Others may give rise to definite symptoms and may lead either to laparotomy for their removal or to laparotomy for more proximate examination and diagnosis.

As a general rule solitary, non-parasitic cysts of the liver occur most frequently in middle or late middle life, 38 of Jones series of 61 cases occurring after the age of thirty-five years. In 4 of the 61 cases age of the patient was not given. Females are more commonly affected, 45 of Jones' series being in women, with sex not indicated in 4 cases. They may occur either with or without evidence of cysts elsewhere in the body, especially in the kidney. In my opinion, however, it is necessary to differentiate the *solitary* non-parasitic cysts of the liver from *multiple* non-parasitic cysts of the same organ. The latter occur nearly always in association with congenital cysts elsewhere and are probably the result, as Moschkowitz[2] clearly points out, of changes which occur in anomalous or aberrant bile ducts in the liver.

[1] Jones, J. F. X. Removal of a retention cyst from the liver. *Ann. Surg.*, 77: 68–89, 1923.

[2] Moschcowitz, E. Non-parasitic cysts (congenital) of the liver, with a study of aberrant bile ducts. *Am. J. M. Sc.*, 131: 674–699, 1906.

The solitary cysts, on the other hand, may be either congenital or acquired as a result of inflammation or direct injury. The majority seem to fall into the group of congenital lesions, some writers even going so far as to include cysts which develop from blood or lymph vessels, and even true multilocular cystadenomata. In my opinion blood vessel or lymph vessel cysts are not, in a strict sence, true liver cysts and will not be considered in this discussion. In like manner multilocular cystadenomata of the liver are considered to be true tumors and in my opinion solitary cysts are not neoplastic. These latter tumors will not, therefore, be considered.

Solitary non-parasitic cysts which are apparently acquired during life, may be due to a number of different causes. Doran[3] believed that the cysts in his patient occurred as a result of trauma. Others have felt that degeneration of the liver from any cause whatever might be followed by cyst formation. Finally solitary cysts may be the result of abnormal retention either as a result of some infectious process or other injury.

Probably most of these cysts are congenital in origin. They may arise in the liver substance itself, which is least common; in the capsule or the sub-capsular parenchymal tissues, which is a very common site of origin; or they may arise extrahepatically, in the suspensory ligament, in the tissues about the gall bladder or the cystic duct, or in the tissues in the transverse fissure on the under surface of the organ.

Cysts arising as a result of trauma are not common. Doran reported one such case in which he believed the cyst to be the result of an injury three years previous to the appearance of symptoms. At operation a large, bile-filled cyst was found. Doran felt that the cyst was the result of the rupture of a bile duct at the time of injury. Bile leakage over a long period of time finally resulted in the formation of a large cyst.

Retention cysts occur as the result either of the blocking off of a normal bile duct or of an aberrant duct, with resulting cyst formation. Such cysts may be deep in the parenchyma of the liver or they may be superficial. In general they do not reach large size.

The fluid contained in the solitary, non-parasitic cysts of the liver varies considerably in character, depending largely upon the type of cyst and its mode of origin. It may be clear and colorless or varying shades of brown or green suggesting bile origin. It may, on the other hand, have a reddish-brown color due to the presence of blood or blood pigment. It may be watery; it may be slightly mucoid and slimy; or it may be thick, dense and sticky. It practically always contains albumin in varying amounts, is usually slightly alkaline in reaction, may contain various pigments of bile or blood origin, and often contains small numbers of cells which may be epithelial, leucocytic, or erythrocytic in type.

The walls of these cysts vary in thickness. In general they are thin and fibrous, but if they are inflamed sclerosis may occur to a marked degree, producing a dense layer that may measure as much as 2 or 2.5 cm. in thickness.

The inner surfaces of these solitary cysts are usually smooth although the cavity of the cyst itself may be irregular due to great numbers of interlacing bands or septa which cross and recross the cyst space. The cavity of such a cyst is often, therefore, a scarred, irregular space, with abundant recesses, depressions, and partial cysts which give to the inner lining the serrated appearance one sometimes finds in degeneration cysts of large uterine leiomyomata.

Under the microscope the inner lining of these cysts consists usually of a single layer of epithelial cells. These cells may be cuboidal, cylindrical, goblet-cell type, or flat and undifferentiated. From the cell type it is not possible to learn anything

[3] Doran, A. *Brit. M. J.*, 2: 1905. (Also *Trans. Roy. Med. & Chir. Soc.*, 87: 1904.)

more concerning the etiology of the cysts than may be learned from other studies. They often suggest, however, that the cyst arises most probably from a bile duct, either a normal duct that became occluded or an anomalous duct that arose as a result of some congenital defect in the development of the liver.

Not infrequently, however, epithelial cells are not found on the inner surface of the cyst. This may be due to the fact that, during the process of distending, epithelial cells previously present failed to keep up with the distention and disappeared or lined only isolated portions of the wall. They may have become so thin from an attempt to cover the greatly stretched wall that they are no longer recognizable as true epithelial cells. Or they may have been absent from the beginning, the cyst having been the result of some inflammatory process which began in fibrous tissue and ultimately produced a large space in this tissue. Such cysts are known to occur in other locations and their lining cells are considered to be either flattened fibrous tissue cells that adapt themselves to a new purpose or endothelial cells from neighboring blood or lymph vessels that might have been injured by the injurious agent that produced the inflammatory process.

The cyst which was removed from the patient in this case consisted of a large, solitary, thin-walled structure measuring 19 × 16 × 18 cm. in size. In both shape and appearance the cyst, as it rested on the table, resembled somewhat a human brain. The outer surfaces of the structure were smooth and shiny except at one pole where the mass had evidently been separated from the tissues of the abdomen. The superficial surfaces appeared to be covered with a thin serous lining beneath which coursed numerous injected blood vessels which ramified and amastomosed abundantly. The cyst was filled with fluid and was very fluctuant. On its under surface and appearing to be an integral part of its wall was an elongated, narrow gall bladder which ended in a severed portion of the cystic duct. The gall bladder contained a very small amount of thick greenish-brown mucoid bile.

On opening the cyst a large amount of thin, clear, greenish, gelatinous fluid gushed forth. This fluid was found to contain albumin but no cells, scolices or hooklets were discovered. The interior of the cyst was composed of a large saccular, thin-walled space which was irregularly crossed and divided into smaller compartments by web-like and strand-like bands of dense connective tissue. The cyst wall was closely adherent to the wall of the gall bladder but at no point were the cavities of the cyst and the gall bladder in actual communication with each other. Grossly, therefore, the cyst was not an abnormal development from the gall bladder itself.

Sections from the cyst wall were studies with the microscope and were found to consist of dense, vascular, congested, chronically inflamed fibrous tissue. The cyst space was lined with no characteristic epithelium in any of the sections studied. The lining cells appeared to be flattened connective tissue cells or perhaps they were endothelial cells which had adapted themselves to this new situation. In one section the wall of the gall bladder was present. It was normal in thickness and its inner surface was lined with normal epithelium. The cyst, therefore, did not appear to be a part of this organ. In my opinion this large cyst arose from an aberrant bile duct which had no connection with the normal biliary system. The absence of typical lining epithelium was not unusual.

Careful examination of the cyst for evidence of parasites or the characteristic scolices or hooklets found in hydatid disease revealed no evidence of any of these parasitic products. The cyst was therefore non-parasitic and in my opinion arose as the result of cystic dilatation of an aberrant bile duct which was probably present as a congenital anomaly.

ILEOSTOMY

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SIDETRACKING of the intestinal contents was first suggested by Littré, in 1710, during the reign of Louis XIV as was recorded by Fontanelle, the historian, to the Académie Royale de Sciences. His idea remained dormant for sixty years when Pillore, a surgeon at Rouen, performed a cecostomy for cancer of the rectum. Duret of Brest, in 1793, performed a successful colostomy from an imperforate anus in a child three days old. With Duret, then, the history of colostomy was begun as stated by Dr. Tilson Dinnick[1] of London. Amussat, a clever and well-known surgeon of Paris, collected the statistics of all the cases from Pillore, in 1776, to which he added his own first case which occurred in 1839. Over a period of sixty-three years, there were 29 cases with a mortality of 20. Four of the cases that survived were for imperforate anus. These were all operated on at Brest. Colostomy, as an operation, was rescued by Amussat from the realm of the occasional and heroic and advanced to its proper place in surgery. He was called to attend the celebrated Professor Broussais in his last illness. He stated, upon witnessing the death of this noted man which was caused by stercoral tympanites produced by rectal carcinoma, that he was determined never again to remain a passive spectator of a death from a similar cause. He employed the axiom of Celsus, "that it is better to employ a doubtful remedy than to condemn a patient to certain death."

Ileostomy has been known since the time of Renault, one hundred and fifty years ago. It is most applicable for temporary drainage using the Witzel technique, or its modification by Charles H. Mayo, or the Hendon technique. For more permanent drainage and sidetracking, the John Young Brown method, utilized by Sistrunk at the Mayo Clinic, may be used. The preferable treatment is the Rankin technique[2] which he used in 1931.

In a certain percentage of chronic ulcerative colitis with complications, we have to resort to surgery. Although it has been recognized that the necessity of surgical interference has gradually diminished with the course of time due to more experience with the vaccine serum, bacteriophage, diet, and earlier recognition of the disease. In these complicated cases, three-fourths of the total number involve the entire colon. Ileostomy, as an operative procedure, is to be considered only as a last resort. It is obligatory that the patient and his family thoroughly understand that the opening is to be a permanent one. This advice also applies to those cases of multiple polyposis or polypoidosis and a certain percentage of cases of diverticula.

Rankin[3] cites the frightful mortality of ileostomy in chronic ulcerative colitis. Eighty-two cases of ileostomy were performed at the Mayo Clinic in the decade 1921 to 1930 for chronic ulcerative colitis resulting in a mortality of 32 early deaths and 15 late ones. Most of these deaths were due to general peritonitis. Rankin[4] stated that "in the acute phase of the disease, the question of operation is debatable." He stated further that "the discouraging results in 33 of the 38 cases (18 operative deaths and 4 later deaths) certainly makes us feel that operative treatment should be undertaken only after the most intensive and thorough medical treatment."

These patients are all very serious risks and the pessimistic attitude of the operating surgeon can be easily appreciated. If

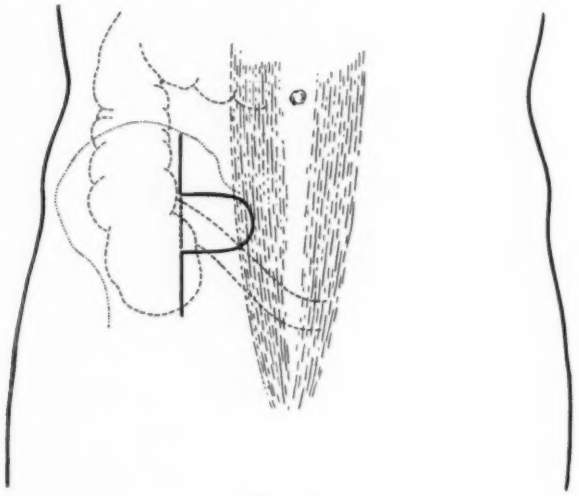

Fig. 1.

we can save the life of an occasional patient by sidetracking the intestinal current as in ileostomy, we should, at least, feel that we are accomplishing some good.

tion of the technique given by Knie, a surgeon in Moscow about 1840. A longitudinal U-shaped incision is made according to Figure 1. Through the skin and the fascia, a tongue-shaped flap is now reflected externally, as in Figure 2, and a straight longitudinal incision is made through the muscle layer and the peritoneum as illustrated in Figure 3. The terminal ileum is brought out through this incision. Generally, this is not very difficult since the patients, on account of their disease, are thin and emaciated. An incision is then made in the mesentery, as in Figure 4, through an avascular area; the proximal portion is at the lower end and the distal portion at the upper end of the incision. The incised mesentery borders of the ileum are sutured through the parietal peritoneum, as in Figure 5, to prevent herniation and obstruction. The parietal peritoneum and muscles are sutured loosely under the overlying ileum. Then, the tongue-shaped flap of skin and fascia are brought through the opening in

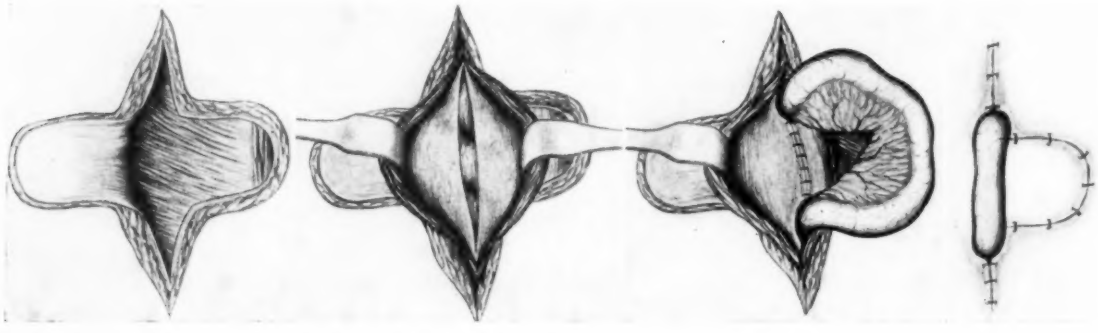

Fig. 2. Fig. 3. Fig. 4. Fig. 5.

My objection to the best known operative procedure, namely that of Rankin, is the fact that clamps are placed across the lumen of the bowel, whose bacterial content is tremendous, and severed by a cautery. It is impossible to prevent peritonitis by this method. I have the same objections to the Lahey technique for the resection of the sigmoid. The operative procedure of ileostomy, that I consider an improvement to the operations, heretofore described, is a modification of the technique given by Knie, a surgeon in Moscow about 1840. A longitudinal U-shaped incision is made according to Figure 1. Through the skin and the fascia, a tongue-shaped flap is now reflected externally, as in Figure 2, and a straight longitudinal incision is made through the muscle layer and the peritoneum as illustrated in Figure 3. The terminal ileum is brought out through this incision. Generally, this is not very difficult since the patients, on account of their disease, are thin and emaciated. An incision is then made in the mesentery, as in Figure 4, through an avascular area; the proximal portion is at the lower end and the distal portion at the upper end of the incision. The incised mesentery borders of the ileum are sutured through the parietal peritoneum, as in Figure 5, to prevent herniation and obstruction. The parietal peritoneum and muscles are sutured loosely under the overlying ileum. Then, the tongue-shaped flap of skin and fascia are brought through the opening in the mesentery and sutured very loosely. After thirty-six hours, the general peritoneal cavity will be walled off so that a division of the overlying ileum can be performed and a Paul's tube inserted in both the proximal and distal ends. Tape, a quarter of an inch in width, can be utilized for ties across the lumen encircling the beveled edge of the Paul's tube. The loss of fluids must be supplanted by clysis in the intravenous route, and for the first three or four days, 4000 to 5000 c.c. of

glucose and saline solution must be given. The sutures in the U-shaped incision are covered with gauze and cotton and sealed off with liberal quantities of collodion avoiding the contamination to which a straight incision would be subjected. The patient must be built up to as healthy a condition as possible. He should be given blood transfusions as needed. After a sufficient time has elapsed, a total colectomy is made, starting at the sigmoid area and using the technique described by me[6] in a recent publication. By using this retrograde colectomy, the distal end of the terminal ileum is the last to be removed.

CONCLUSIONS

1. Rankin[5] has furnished ample evidence that surgery has failed to be of any great benefit in many cases of acute ulcerative colitis and it is only in the chronic complicated cases which, for over a long period of time, have failed to derive any benefit from medical care.

2. Ileostomy should be performed only with the full understanding of the patient and his family that the opening is to be a permanent one (ileostomy).

3. In multiple polyposis or polypoidosis cases, the operative outlook is much brighter. In those cases of multiple diverticula, which are complicated by a recent diverticulitis, the acute symptoms must have subsided for some time before any operative procedure is even to be considered.

4. In those longstanding cases of chronic ulcerative colitis with complications, and those cases of diverticula with diverticulitis, and even in some of the cases of multiple polyposis or polypoidosis, we have a tremendous influx of pathogenic bacteria both in the colon and in the terminal ileum. It is for this latter reason that I have designed this asceptic technique for performing a permanent ileostomy.

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CONGENITAL ABSENCE OF THE GALL BLADDER

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"COMPLETE absence of the gall bladder in the human is of very rare occurrence, although the condition has been described." This quotation, from one of the latest and most modern text-book on gall-bladder disease,[33] is substantiated by the scarcity with which this condition is reported in the literature. Since the gall bladder is normally absent in certain animals and may be present and at other times be absent in some animals of the same species, its absence in the human has been considered atavistic by some writers. Congenital absence of the gall bladder is frequently associated with other defects and anomalies, such as anomalies of the ears and palate, supernumerary spleen, megalocolon, imperforated anus, etc.

Courvoisier was the first to collect 25 cases of gall-bladder absence in 1890.[34] In a series of 7919 autopsies reported by T. C. Smith and R. T. Ball,[30] Mentzer and Nagel,[35] L. C. Knox (quoted by W. Lintz,[37] and Kehr, there were 6 cases of congenital absence of the gall bladder. J. O. Bower[36] reported a case of congenital absence of the gall bladder and tabulated 67 other cases that were reported up to that time by other surgeons.

My personal experience with this peculiar anatomical anomaly is limited to one case, in which the absence of the gall bladder was definitely established by operative and postmortem findings. This prompted me to make a careful search of the literature for additional case reports. Twenty-seven additional cases of congenital absence of the gall bladder were reported in the literature from the time that Bower published his paper until August, 1933. These cases, added to those already reported by Bower and including my own case, which I am about to report, make a total of 96 cases of congenital absence of the gall bladder, reported up to the present writing.

Disregarding its infrequent occurrence, it should be given careful consideration whenever cholecystographic examinations are made or during the performance of an operation for supposed gall-bladder disease. Failure to obtain distinct gall-bladder visualization after the administration of the dye, which is usually considered very strong evidence of gall-bladder disease, may, in rare instances, be due either to a rudimentary or a totally absent gall bladder.

In some of this group of 96 collected cases, positive evidence of absence of gall bladder is missing. The diagnosis was made by the surgeons on the operating table who reported inability to find the gall bladder. One must bear in mind that such inability to find the viscus does not necessarily mean that the gall bladder is not present. In some instances, the inability to find it may be due to extensive adhesions and exudative deposits covering the gall bladder, as a result of recent inflammatory reaction around that viscus, or it may be due to an atrophy of that organ, to the size of a small fibrous duct as a result of frequent attacks of cholecystitis.

Intrahepatic location of the gall bladder, while of extremely rare occurrence, should also be given very careful consideration whenever the gall bladder cannot be found at its usual location at the time of operation, since it is very easy to misinterpret a complete intrahepatic gall

bladder for a congenital absence of the same organ. It is possible that some of the cases reported under the heading of "absence of gall bladder," may have been instances of complete intrahepatic location of that organ. Several such reports have appeared in the literature, in which a supposedly absent gall bladder was subsequently proved, either by reoperation or by *x*-ray, to be an intrahepatic gall bladder.

In one case in which the operative report was "absence of the gall bladder," subsequent *x*-ray examination showed a sausage-shaped shadow in the region of the gall bladder. In another case thorough search by two competent surgeons failed to reveal the gall bladder. A normal appendix was removed and the abdomen was closed.[37] Two weeks following the operation, symptoms of gall-bladder disease recurred and the diagnosis of choledochoduodenitis or pancreatitis was made. The Lyon-Meltzer test two weeks after readmission showed three different colored specimens of bile: (a) golden yellow, (b) dark green bile with much mucus and (c) light yellow fluid. Congenital absence of the gall bladder is questioned by the surgeon in this case since one of the specimens of fluid obtained through the duodenal tube, following the operation, proved to be B bile, which is supposed to come from the gall bladder. Either the theory advanced by many observers that B bile comes only from the gall bladder is incorrect, or else this patient may have had her gall bladder imbedded very deeply in the liver and thus overlooked during the operation.

Four cases of intrahepatic gall bladders are reported by Kehr.[19] In one of these, the diagnosis of absence of the gall bladder was made at operation and the abdomen was closed, but the attacks of colic, requiring frequent administration of morphine, recurred. Two years later this patient was reoperated on, the liver was incised and a deeply imbedded gall bladder, containing two stones, was found. In 3 other patients operated on subsequently, in which the gall bladder was not visible at its usual location at the operation, the possibility of an intrahepatic gall bladder was considered, and benefiting by the experience of his first case, he incised the liver and a gall bladder containing many stones, deep in the liver substance, was found.

FIG. 1. Specimen, showing (A) common duct, (B) left hepatic duct, (C) right hepatic duct, (D) ampulla of Vater.

Dévé,[6] in a postmortem examination of 130 infant's livers, found 11 instances of intrahepatic location of the gall bladder. In 3 of these the gall bladder was entirely imbedded in the liver structure and in 8, only the fundus was imbedded.

The total number of cases included in this report were 96; 28 were males and 36 were females. In 32, the sex was not mentioned. The diagnosis of congenital absence was made by autopsy and operation in 65 cases, by operative findings only in 22 cases, and in 9 the means of arriving at a diagnosis is not stated. (Table 1.) Thirty-one of these patients were infants less than one year old. A large proportion

TABLE I

Total number of cases	96
Number of cases collected by Bower	67
Bower's own case	1
Number of cases collected from the literature since the publication of Bower's paper and not included in his series	27
Author's case	1
Total	96
Number of males	28
Number of females	36
Not mentioned	32
Diagnosis:	
(a) By autopsy and operation	65
(b) By operative findings	22
(c) Not mentioned	9

of these infants showed symptoms of biliary disease, such as jaundice, profuse melena, bleeding from navel, marked pallor, clay-colored stools, etc., and the anomaly was discovered when the operation for the relief of this condition was performed. In a few the condition was discovered by postmortem examination. In most of these infants the gall bladder and the extrahepatic ducts were absent. (Table II.)

TABLE II

Foetus	2
One week	8
Two weeks	2
One month	3
Two months	5
Three months	4
Four months	3
Six months	3
Eight months	1
One year	2
Two years	1

Thirty-eight cases included in this report showed absence of the gall bladder. In 16 of these cases, no mention is made as to the presence or absence of extrahepatic ducts. In 26, the gall bladder and cystic duct were absent. Fifteen showed absence of gall bladder and extrahepatic ducts. Two showed no gall bladder, one common hepatic duct leading directly into the duodenum. One showed no gall bladder, no cystic or common duct; each had two separate hepatic ducts which opened directly into the duodenum. Three showed no gall bladder and no cystic duct, one common hepatic duct. In 6, only rudimentary gall bladders were found. Two showed no gall bladder and no cystic duct, two right and one left hepatic duct in each case. Two showed a small rudimentary intrahepatic gall bladder, and in one case there was absence of the gall bladder, cystic and common duct and the left lobe of liver. (Table III.)

TABLE III

No gall bladder	38
No gall bladder and cystic duct	26
No gall bladder or extrahepatic or common ducts	15
No gall bladder, two right and one left hepatic duct	2
No gall bladder, one common hepatic duct leading directly into duodenum	2
No gall bladder, no cystic or common duct; two separate hepatic ducts	1
Small intrahepatic gall bladder	2
No gall bladder, no cystic duct; one common hepatic duct	3
Rudimentary gall bladder	6
Absence of gall bladder, cystic and common duct together with an absence of the left lobe of the liver (Bower's case)	1
Totals	96

In 16 of the 38 cases listed as "no gall bladder," no mention is made as to the presence or absence of extrahepatic ducts.

CASE REPORT

R. L., white, female, aged fifty-three, occupation, housewife; nativity, Russia; was admitted to the Newark Beth Israel Hospital with a provisional diagnosis of cholecystitis and cholelithiasis. Her chief complaint was pain at the right upper quadrant radiating to the back. The pain was preceded by belching and vomiting after eating.

Present History. Patient states that ten years ago she had severe pain in epigastrium which lasted several hours. It left her for several months, then these attacks began to come on on an average of every six months. They were associated with vomiting and much belching. During the last three months these attacks would come on every two days and lately she was getting several attacks a day. She has consulted several doctors for these attacks. She is afraid to eat for fear of precipitating an attack. During the last year she experienced considerable weakness in her legs and, recently, she can hardly walk.

Past History. None of childhood diseases. Typhus fever thirty-five years ago. Menopause one and one-half years ago. Marital:

Husband living and well. Six children alive and well. One miscarriage following the typhus fever. Had much trouble during one twin delivery. Habit: Bowels constipated; takes cathartics regularly. Was never operated on.

Physical Examination. Essentially negative. Moderately emaciated person, appeared rather weak. The heart sounds are of good quality, note and rhythm. No murmurs. Chest symmetrical; no râles. Some tenderness over the right hypochondriac region, particularly on deep pressure. No palpable masses. No abnormal reflexes present. There was a marked rectocele and cystocele. Uterus somewhat retroverted.

Patient was operated on on November 19, 1929, with the following findings: Gall bladder and cystic duct absent. The common duct is markedly dilated and bifurcates into the hepatics. The lower end of the common duct is filled with soft stones and inspissated bile. Clear bile flowed from the upper end of the duct.

Operative Procedure. Abdomen opened and explored through high right rectus incision. The common duct was identified and traced down to its insertion behind the duodenum. It was opened an inch above its entrance into the duodenum and the stones removed. A probe passed down into the duodenum without meeting any obstruction. A T-tube was inserted and the incision in the duct was closed around the tube by interrupted fine chromic gut sutures. Abdomen was closed in layer sutures.

Postoperative Course. Patient returned from the operating room at 12:15 P.M. on November 19, 1929. The usual immediate postoperative treatment was administered. At 2:00 P.M. the patient complained of marked abdominal pain and was extremely restless. At 2:30 P.M. ¼ grain of morphine was administered, followed by some relief; but at 6:30 P.M. patient began to complain again of severe pain, moaning constantly and extremely restless, requiring another hypodermic of morphine for relief. The pulse at that time was 100, respiration 20. There was a profuse discharge of a dark bloody-looking fluid from the wound. She slowly went into a very profound state of shock at 10:00 P.M., nine hours after the operation. Pulse was 110, rather small and at times imperceptible. The usual treatment for shock was instituted, hypodermic clysis of 900 c.c. of glucose was administered, and the foot of the bed was elevated. The question of internal hemorrhage was considered, but the patient's general appearance was not that of exsanguination.

At 11:45 P.M. 750 c.c. of blood were given by the Lindeman method. This was followed by a temporary improvement of her condition. The quality of the pulse improved considerably, the rate going down from 110 to 90. During that time the patient was in a semi-comatose state, did not respond to any questions. Her respirations were shallow but were almost at a normal rate until the following day when the rate was increased to 40, then 50, and almost 60, before death.

Blood pressure at the time of the transfusion was 90/70. External heat was continued. Digalin and atrophine was administered. Thromboplastine of 10 c.c. was also given.

The beneficial effect of the transfusion was of short duration. At 1:45 A.M., two hours after transfusion, pulse again became weak and almost imperceptible. Temperature was 102.8°F.; pulse 110; respirations 22.

At 4:45 A.M. (sixteen hours after the operation) the patient was catheterized and only about 1½ ounces of urine obtained, in spite of the fact that up to that time the patient's intake of fluid was over 2,000 c.c., including the blood transfusion.

At 5:15 A.M. (same day) another clysis of 500 c.c. of glucose was given. There was some abdominal distention, continual eructation of gas from stomach, lips and tongue very dry. Pulse still very weak and almost imperceptible.

At 7:15 A.M. caffein sodium benzoate of 7½ grains, atrophine, 1/100 grain, digalin, minums 30 were administered. Respiration at that time was 44.

Another transfusion of 500 c.c. of blood and 700 c.c. of glucose was administered at 9:00 A.M. and this was followed by a continuous intravenous administration of glucose. The patient again seemed to have responded to the transfusion. The pulse was 120, but of very good quality. Respirations at 11:30 A.M. were 42. About 1:00 P.M. (same day) the patient vomited a small amount of watery fluid. At 4:00 P.M. the pulse again became very weak and thready and again imperceptible. The temperature rose to 103.2°F., respirations 42.

Another 1100 c.c. of glucose were administered at 6:00 P.M., followed by a third transfusion at 6:30 P.M. Patient again responded to transfusion. Pulse 114, of fairly good quality.

A dark-bloody discharge from the wound continued.

Patient was again catheterized at this time and only 6 ounces of urine obtained, making a total of 7½ ounces during the thirty-two hours following the operation. The total intake of fluid during that time was 6000 c.c. The urinalysis showed the following: reaction, acid; specific gravity, 1024; albumin, 2 plus; glucose, negative; few hyaline and granular casts.

At 9:00 P.M. patient appeared a little brighter, moaning continuously and quite restless. Intravenous administration of glucose was again started and digalin minums 30, was administered.

At 12:30 P.M. the effect of the transfusion wore off, the pulse again was imperceptible, respiration 60, rapid and labored. Patient lapsed into a state of coma. Pulse rate not obtained, temperature rose to 104°F., discharge from the wound still continued. No more urine was obtained. The total output during the forty hours was 7½ ounces. The patient's condition became progressively worse and she died at 3:45 A.M. (about forty hours after the operation).

Report of Autopsy: Pathologist: Dr. Asher Yaguda.

External Examination: Body is that of a well-developed, well-nourished white female adult. There is an incision over the right rectus muscle extending from the xyphoid process to just below the umbilicus. This incision is sewed up by deep silkworm gut sutures and skin clips, and from the upper end of the incision protrudes one cigarette drain and one piece of rubber tubing. There is moderate lividity of the extremities and a mottling lividity of the dependent portions of the body. The right cubital fossa has been opened for intravenous work and the vein exposed. There are no other external evidences of injury.

Internal Examination: Peritoneal Cavity: Upon opening up the layers of the wound, there is seen to be an area of slough and infection along the course of the drains. The muscle is infected and decomposed. Peritoneal cavity contains some free-bile-stained fluid. Peritoneum has lost its glistening appearance and is covered with a yellowish exudate which agglutinates the loops of gut. In the pelvis is a collection of fluid which is bile stained, dark brown in color. The omentum is injected.

Pleural Cavities: No free fluid in either pleural cavity.

Pericardial Sac: Not dilated. Pericardium smooth and glistening.

Heart: Tricuspid admits just three fingers, is not dilated. Heart muscle is pale, thin and fatty. Endocardium is clear. Pulmonary artery is grossly negative. Left heart muscle is soft, friable and thin, pale in color and has a boiled appearance. The mitral admits two fingers, and there is no evidence of any endocarditis. Aorta is grossly negative.

Left Lung: There is a marked bogginess of the entire lower lobe and posterior and lateral portions of the upper lobe due to congestion and edema. The rest of the lung is emphysematous.

Right Lung: Same as left.

Spleen: Not enlarged. On section slightly mushy in consistency. Otherwise negative.

Left Kidney: Small. Cortex swollen, edematous, pale. Pelvis of the kidney is grossly negative. There is some slight injection of the vena recti, but this is not marked.

Right Kidney: Same as left.

Adrenals: Show some degeneration of the medulla. Otherwise negative.

Gastrointestinal Tract: Stomach dilated. Contains some bile-stained fluid. There is no evidence of any ulcer. Duodenum, mucosa is covered with mucus and a yellowish fecal appearing substance. The rest of the intestine, except for the exudate and injection of the serosa, is grossly negative. Common duct approached without difficulty through the papilla of Vater. Upon opening up the common duct, a rubber tube is seen to be in the common duct and extends into the duodenal and pancreatic portion of the common duct. On opening the duct in its entire extent, there is seen to be an opening on the anterior surface just above the duodenum, through which a T-shaped rubber tube has been inserted. The common duct is easily followed to its dividing line into right and left hepatic ducts. The right hepatic duct is dilated and can be dissected back into the liver, as can the left. The entrance to the cystic duct cannot be found. There apparently is no cystic duct.

Gall Bladder: Also cannot be found. The gall-bladder fossa in the liver contains some hemorrhagic material which is seen to be blood clot and fibrin. No evidence of the gall bladder can be made out within the liver substance.

Pancreas: The pancreas has been dissected away from the common duct on its inner aspect, and there is slight digestion of tissues in this location at the head of the pancreas. The rest

of the pancreas appears to be normal grossly with the exception that the lobules of pancreatic parenchyma are separated widely by fat.

Aorta: Grossly negative.

Liver: Small. Appears to be fatty, but otherwise negative except for a cloudy swelling.

Uterus, Tubes and Ovaries: Grossly negative.

Bladder: Negative.

Cause of Death: Generalized peritonitis following operation for stone in common duct.

Secondary or Terminal Lesions: Pancreatitis head of pancreas. Toxic myocardial degeneration. Acute pulmonary congestion and edema. Cloudy swelling of liver, spleen and kidneys.

Historical Landmarks: Absence of gall bladder and cyst duct (congenital).

DISCUSSION

The two most outstanding points of interest in this case are: (a) the congenital absence of the gall bladder and cystic duct with marked dilatation of the common duct, definitely proved by operation and autopsy; (b) a most unusual postoperative course, manifested by severe and persistent shock, resisting all efforts to combat it, ending fatally within forty hours.

In reviewing the postoperative course it is shown that the patient went into a profound shock soon after the operation and did not respond to the various measures instituted to combat it. Throughout the operation the patient's condition was favorable and she did not show any evidence of shock until nine hours later. The question of internal hemorrhage was considered, but the patient's general appearance was not that of exsanguination. Repeated large transfusions failed to show any appreciable effect on the patient's condition. When the patient became pulseless and semicomatose and almost moribund, a transfusion would be followed by marked improvement in the quality of the pulse and a decided change in her entire appearance. She would become mentally alert, respond to all questions quite rationally and the skin would become warm and of good color. Respirations were almost normal, but this change was of very short duration. Within two or three hours patient again went into general collapse and another transfusion was given. The total fluid intake, from the time of operation until her death, was 6000 c.c., of which 1750 were blood and the remaining 4250 were 5 per cent glucose. The urinary output throughout that time was only 7½ ounces, showing a distinct suppression of kidney function.

Although the autopsy record gives the cause of death as "generalized peritonitis following operation for stone in the common duct," which I have no doubt may have been the major cause, I do feel, however, that the clinical behavior in this case, following the operation, justifies the assumption that liver damage followed by hepatic and renal exhaustion of the type so frequently described previously in the literature, may have acted as a contributing cause.

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[For Remainder of References see p. 308]

MESENTERIC THROMBOSIS

(ARTERIAL AND VENOUS TYPES AS SEPARATE CLINICAL ENTITIES) A CLINICAL AND EXPERIMENTAL STUDY

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INTRODUCTION

MESENTERIC thrombosis forms only a small percentage of the cases ordinarily listed under the Intestinal Obstruction heading. McIver reported its occurrence as about 3 per cent in a collection of 335 cases of intestinal obstruction.[15] The seriousness of the disease, however, and certain of its fundamentals, make constructive consideration more than worth while.

Here and there in the journals and texts, one may find an occasional mention of separate thrombosis of the mesenteric veins and arteries. We have been unable to find, however, a clear-cut discourse on the differentiation of venous and arterial mesenteric thrombosis as separate clinical entities. We feel that a sharper descriptive differentiation should be incorporated in the texts, and that a wider dissemination of knowledge in the current journals regarding a differentiation is not out of place.

It would seem that the words "mesenteric thrombosis" immediately bring to the minds of most surgeons the rapidly and consistently fatal arterial syndrome which is familiar to all of us and which is generally recognized as being only rarely diagnosed preoperatively. The fact that the possibility of an occurrence of a slowly progressing condition of mesenteric thrombosis which in the early stages is most thoroughly amenable to surgery does not register in the minds of the profession generally, is to a certain extent responsible for the inaccuracy of diagnosis and high mortality connected with the condition.

Our present interest in this condition was initiated by a human case, in which one of us operated in 1933; and we set about in October, 1933 to carry out some experiments on intestinal obstruction,[8] including work directed toward a more clear-cut differentiation of two types of mesenteric thrombosis.

Since the condition and clinical picture of arterial mesenteric thrombosis is well described in the literature, our remarks here are confined chiefly to the less clearly understood venous type. Human cases and experimental studies are presented and reviewed in an attempt to more definitely catalogue relevant diagnostic and therapeutic considerations.

ETIOLOGICAL CONSIDERATIONS

It is logical to assume that the same general elements which play a part in thrombus formation in any of the vessels of the human body, play a similar rôle in thrombus formation in mesenteric vessels. We can readily recall in a general way the final processes of clot formation by the thromboplastic blood elements, together with predisposing physical factors such as the age element, emboli, trauma, and a sluggishness of the blood stream, and the chemical and bacterial factors such as toxin production and absorption. And considering the intestinal tract in the light of these elements, there seem to be a number of factors which should, theoretically, *predispose to a more frequent thrombosis in the venous apparatus of the mesentery*, than in the arterial. For example, the flow of the veins from the great scavenger tube, the intestinal tract, is subject at least to some extent to the absorption of various

intestinal toxins. The decreased muscular resistance of the capillary walls as compared to those of the arterioles, the decreased velocity of blood flow, and the increased wall fragility, would tend to make the venous apparatus somewhat more susceptible.

Specific conditions which we feel may cause primary mesenteric venous thrombosis without arterial thrombosis are as follows:

1. Intussusception with sufficient pressure to obliterate the venous return but not the arterial flow.
2. Volvulus with the same pressure conditions.
3. Absorption of non-bacterial toxin or chemicals from the intestinal tract.[12]
4. General physical debility and upset physiology which goes with acute infectious "fevers."
5. Trauma.[18]
6. Paralytic ileus or other conditions giving a high intraintestinal gas pressure.
7. Infective or other emboli.
8. Bacterial or non-bacterial *enteritis, chronic or acute.*

SYMPTOMS OF VENOUS MESENTERIC THROMBOSIS

Since venous mesenteric thrombosis may occur as a complication of various conditions such as those just mentioned, it is obvious that the picture may be complicated by various elements of pathology. Generally speaking however, except in that type of venous mesenteric thrombosis initiated for example by sudden volvulus or intussusception, the onset and course would seem to be relatively slow, and initiated by pains of a moderate collicky nature. Two of our case histories here reported and two other similar histories of cases found in the recent literature, are particularly interesting as regards the several days' duration of symptoms before the victims seem to have given themselves particularly serious consideration.[5,12] Another striking symptom in the syndrome of this disease is the disproportion of the abdominal tenderness and the duration of symptoms, to the abdominal rigidity. The abdomen is usually widely and definitely tender to deep palpation and the abdominal rigidity is not nearly as marked as one might expect it to be when the possible duration of the complaint and the definite tenderness are considered. The normal leucocyte relationship is only moderately disturbed in the early stages, but is markedly distorted later. An engorged intestinal wall may or may not be felt as a palpable mass. *Occult blood is uniformly present in the stool and bowel movements continue,* dark and soft to liquid in nature. Dark brown "coffee ground" like vomitus may supervene. The temperature range in the uncomplicated picture is low. Intestinal "fluid levels" are usually absent. *Unless the possibility of the disease is kept in mind it will be invariably very difficult to recognize it as a definite surgical condition in its earlier stages when surgery should offer a very excellent prognosis.* Intestinal obstruction, appendicitis, peritonitis, peptic ulcer, lead colic, "simple" enteritis, cholecystitis, gastritis, typhoid fever, are among the diseases most likely to be confused by the expert.

DIFFERENTIAL DIAGNOSIS

Arterial Thrombosis: rapidly fatal; marked toxicity within eight to eighteen hours; onset usually sudden and rapidly progressive; shock of greater degree.

Intestinal Obstruction without Gangrene: pain of collicky nature progressive in character; vomiting early or late; abdomen becoming somewhat more rigid at least eventually; fluid levels present. Increased peristalsis followed later by absence; bowel movements unsatisfactory.

Obstruction with Gangrene: rapid and progressive toxicity when absorption from gangrenous tissue is taking place. Picture similar to arterial mesenteric thrombosis. Other signs of intestinal obstruction varying and depending on condition.

Infectious Diseases: usually an onset with more fever and malaise.

Food Poisoning: History, nausea, diarrhea, shock.

"Simple" Enteritis: Usually diarrhea, cramps more collicky, history of food indiscretion usually more definite.

PROGNOSIS

Conjecturally speaking, the human mortality, in a venous thrombosis involving even several inches of bowel, may not be over 50 to 70 per cent, without surgical interference. This condition may explain some of those puzzling cases in which we find at the operating table old abdominal adhesions, with no attendant clear-cut explanation for their presence being available. Osler once said the prognosis of the patient with mesenteric thrombosis was perhaps as good without as with surgery and, surprisingly, gave the non-operated mortality at 50 per cent. He apparently however did not differentiate in detail between the two types. Arterial thrombosis sufficiently extensive to give any gangrene of any segment, will create a mortality of practically 100 per cent without surgery and, at present, over 50 per cent with surgical interference. That the venous condition when at all extensive is surgical seems unquestionable. The chief value of one's familiarizing himself more thoroughly with the disease would seem to be in enhancing one's ability to recognize it as a clinical entity earlier in its course before progressive thrombosis has initiated a constitutional condition unfavorable to general constitutional recovery, or, as in case 1, a condition predisposing to a progressive continued thrombosis after gut resection. With early recognition and proper treatment the mortality should be considerably lower than is the present mortality of "mesenteric thrombosis." Too, the morbidity in the cases which recover should be definitely more satisfactory.

MORBID ANATOMY

Complete venous thrombosis gives marked congestion of the gut wall tissues producing grossly an edematous dark bluish soggy mass which at first glance would seem devitalized beyond possible

TABLE 1
PURE VENOUS LIGATION
(By method of Figure 1)
Dogs

No.	Date	Area	Result
1	10–3–33	8 cm. low ileum	In an area this small, venous ligation by our method does not as a rule produce much infarction. Recovery in dogs is almost 100% in an area thus limited.
2	10–6–34 11 A. M.	15 cm. low ileum	Killed at 5 hours. Dark edematous discoloration of entire segment. Arteries fully patent. Muscular reaction satisfactory.
3	10–6–34	15 cm. low ileum	Killed, 72 hours. Gut infarcted. Viable. Muscular reaction sluggish but present. Dog good general condition. All arteries fully patent. No peritonitis.
4	11–16–34	15 cm. low ileum	Died, 4½ days. Obstruction cause of death. Gut area infarcted but viable. Local peritonitis with adhesion produced obstruction.
5	11–16–34	15 cm. low ileum	Killed, 2 months. General condition good. Clinical recovery complete. Many adhesions at local site.
6	2–17–34	15 cm. low ileum	Killed one month. Complete clinical recovery. Many local adhesions.
7	9–23–34	25 cm. low ileum	Killed 3 wks. Had recovered completely. Nutrition however somewhat impaired, from, apparently, partial obstruction. Many local adhesions. See Fig. 5.
8	9–23–34	20 cm. low ileum	Killed 2½ weeks. Had recovered completely and was well nourished. Many local adhesions.

recovery. *The muscular structure however does remain viable* and muscular contraction, though sluggish, can be produced. The concomitant arterial system remains patent with a surprising persistency. The lymphatics partially carry on the function of the venous return. The gut wall usually remains intact enough to prevent at least an extensive bacterial peritonitis. The hemorrhagically enfarcted area, remaining supplied by impaired but functioning arterial blood flow, may adjust itself by establishing a fibrotic gut wall which requires only a scanty blood supply with some collateral venous return at times being established through the omentum (see Fig. 5); or the impaired gut tissue may deteriorate to such an extent that sufficient toxin is liberated, in conjunction with bacteria or not, to produce general vital organ degeneration and death. Local abscess formation may occur. Extensive adhesions form about the involved gut area during the process of recovery. Intestinal obstruction, partial or complete, may result from adhesion formation. (See Table 1 and Fig. 5.)

HUMAN CASE REPORTS

Case 1. Case No. 61773. Robert B. Green Hospital. Surgeon: J. K. Donaldson.*

Mexican female, housewife, aged sixty, admitted to Robt. B. Green Hospital, 4 P. M. March 25, 1933.

Present Illness. Seven days ago patient was seized during afternoon with rather sudden severe pains in abdomen. No history of undue exercise or trauma could be elicited. Pain rather promptly subsided to some extent *but continued intermittently, as pains of moderate colicky nature. Bowels since onset moved about once a day with some increased fluidity and "darkened color."* Some nourishment had been taken all during the seven days prior to entrance. Patient's customary diet was of rather large quantity and was composed chiefly of spicy Mexican foods. No other history of unusual dietary indiscretion was obtained. Emesis of a "moderate" quantity of dark fluid had occurred two to three times in twenty-four hours prior to admittance. A moderate dosage of a patented laxative was apparently taken early in the course of the disease.

Family, Past and *Social History:* All apparently irrelevant.

Physical Findings. Rather obese woman appearing somewhat younger than stated age; weight 160, height 5 ft. 5 in. The patient could walk but with effort, upon admittance, and seemed generally in poor condition. The skin was dry, cool, temperature 100.4°F., pulse 128, respiration 36. An emesis of dark brown fluid was recorded upon the chart shortly after admittance at 4:00 P. M. Examination revealed *general definite abdominal tenderness to deep palpation, more marked right half, with only very moderate generalized rigidity.* Panniculus of abdomen was quite thick and no mass could be felt. No peristalsis was heard. Abdomen moderately distended. There was essentially no rebound tenderness. The heart sounds were somewhat distant and free from murmurs. P. 2 was moderately accentuated. Blood pressure was 160/105. All reflexes and other parts of physical examination including a careful check of lungs were essentially negative or irrelevant.

March 25, 1933. Blood Count: Total white cells 36,650; total neutrophiles 90; segmented 80; stabs 6; juveniles 4; Eosinophiles none; basophiles none; monocytes 2; myelocytes none.

Urine Analysis: Specific gravity: "insufficient."

Albumin: One plus.

Sugar: Trace.

Microscopic: "Three plus hyaline cast with occasional pus cell."

Roentgenological Examination. "No evidence of gas distention or fluid levels in small bowel."

Operation. Exploration was done after about three and one-half hours of preoperative therapy had been given. Free, thin, bloody fluid was present in the abdominal cavity in considerable quantity. A dark bluish, soggy gangrenous looking area of lower ileum about 24 inches in length was exposed. No direct obstruction of any sort was apparent. An immediate diagnosis of mesenteric thrombosis was evident. Immediate resection was done. In cutting through the mesenteric vessels of the involved area a free arterial, but no venous bleeding would occur. There was complete thrombosis of all the venous radicals of the

* Operating as an Associate on Surgical Service No. 3.

involved area, but no thrombosis of the concomitant arterial system was seen. The two ends of gut were rapidly brought out without anastomosis and the abdomen closed as rapidly as possible. We resected the gut for a distance of about 4 inches on each side of the grossly involved area.

The patient left the operating table in a general state of rather severe shock, recovered to some extent, but expired at 10:45 P.M. March 26, 1933, approximately twenty-eight hours after operation.

Autopsy (Dr. Todd). Grossly all the vital organs were essentially negative except for moderate congestion and definite "softening" of the cardiac musculature. Heart valves were perfectly normal. There was an area where venous thrombosis had progressed after operation and which involved approximately 6 inches of ileum on each of the ends from which the originally involved gut had been removed. An antemortem clot, beginning to impinge into the lumen of the main trunk of the superior mesenteric vein, was demonstrable. The mesenteric arterial system in both the resected specimen and the retained mesentery was grossly entirely free from arterial thrombosis. Microscopic study of the organs was not done.

Case II. No. 61531: Santa Rosa Hospital. Surgeon: Dr. Homer T. Wilson.

Male, aged forty-nine, broker, admitted December 15, 1933 giving history of having eaten, *about three weeks previous*, on a Saturday afternoon, a rather large quantity of Mexican food. About 10 A.M. the next day he had marked diarrhea and pain in the abdomen. He was ambulatory, did not go to a doctor, took paregoric and milk of magnesia. Diarrhea continued off and on for *three weeks during the last two of which he took paregoric and milk of magnesia every day*. No vomiting. Remainder of history irrelevent or negative.

Physical Findings. Excerpt from interne's admittance record is as follows: "Entire abdomen has pressure tenderness particularly in both lower quadrants, rigidity in both lower quadrants, no masses, no hernia, McBurney's point more tender, all reflex signs indicate appendicitis."

Fluoroscopic Examination. Barium enema December 16, 1933. Fluoroscopic: "Excessive gas in lower bowel. No other abnormalities apparent between hepatic flexure and rectum."

Laboratory Examination. December 15, 1933. Urine analysis: "Several coarse granular casts with occasional pus and red blood cell."

Blood count: R. B. C. 4,700,000; Total white 12,750; H. G. 80; polys. 87; S. L. 7; L. L. 6; eos. none; trans. none.

December 16, 1933. Blood count: total 17,850; polys. 95; S. L. 6; L. L. 4; eos. none; trans. none.

The morning of December 16, 1933, one day after admittance, the patient was being prepared for surgery when he went into a state of acute collapse and general sustaintive measures were instituted. Could not be got in condition for surgical exploration and expired December 16, 1933 at 11:15 P. M.

Autopsy (Dr. J. M. Moore). One of the pathologists doing the autopsy, being aware of our previous case and interest in this condition, notified us after the abdomen had been opened, and one of us was present (Dr. Wilson's courtesy) at the post mortem. Excerpt from the records of Dr. J. M. Moore follow:

"There was some free, thin bloody fluid present in the peritoneal cavity and the small intestine was distended. The small gut appeared dark blue, edematous, and semi-gangrenous from Treitz's ligament to splenic flexure." "Grossly all vital organs were negative except for congestion." Rectum: Bloody, black, fecal discharge. Microscopic: The liver showed marked parenchymatous change with much fatty degeneration. Practically no intact liver cells found. Heart: Considerable degenerative change manifested as cloudy swelling and fatty necrosis. Involved bowel: Section shows only edema and necrosis; not able to make out any cellular detail. Kidneys: Acute parenchymatous degeneration. The superior mesenteric vein showed an antemortem thrombosis extending throughout its branches up and through its main stem almost to the portal. The superior mesenteric artery and its branches did not show evidence of antemortem clot. The condition was one of extensive and pure venous thrombosis.

Case III. Nix Hospital Case: 3837. Surgeon: Dr. Harry Mc C. Johnson.

Male, retired "financier," aged sixty-eight, admitted March 21, 1933.

History. History of digestive disturbances suggestive of cholecystitis extending over a period of several years with particular reference to collicky pains in left upper quadrant. These were some worse for three months prior to operation. G. I. series had revealed no ab-

normality. Typical history of frequency, difficulty of urination suggestive of hypertrophic prostatitis. Three ounce residual urine. History including recent dietary regime, exercise and trauma, recorded as otherwise negative or irrelevant. March 22, 1933, Dr. Mc C. Johnson did a transurethral prostatic resection uneventfully. The patient left the table in good condition. On awakening from anesthetic several hours afterward the patient began to complain of some pain in the abdomen of colicky nature and vomited dark brownish colored material, which vomitus was repeated later a number of times. The morning of March 24, 1933 the patient went into a condition described as general shock and all measures being of no avail, he died at 8:30 P. M.

Past History. Three years ago cystitis, prostatic abscess, epididymitis, "cured" in three to four months. History otherwise seemingly irrelevant.

Physical Findings. Weight 160. Height 5 ft. 6 in. Appeared somewhat younger than stated age. Dr. Mc C. Johnson thought, when the post operative syndrome developed, of hemorrhage from the operation; but investigation revealed that no such occurrence was taking place. There was some distention of the abdomen. *There was not as much rigidity as one would ordinarily expect in proportion to the generalized tenderness which was present to deep palpation.* No masses were felt.

Laboratory Examination. March 21, 1933. R. B. C. 5,000,000; H. G. 96; color index 0.9; total leuco. 12,400; S. F. 64; polys. 66; lymph. 31; stabs 2.

March 24, 1933. H. G. 96; R. B. C. 3,260,000; tot. leuco 8250; Y. F. 4; stabs 36; S. F. 55; polys. 95; lymph. 5; mono. 3.

Urine analysis, essentially negative.

Cystoscopic: Diverticulum thumb size superior and posterior part of the bladder, right.

Autopsy (Excerpt) (Dr. Todd). Abdominal cavity presents large amount of sero-sanguinous fluid in which there are floating many small particles of a fibroplastic exudate. This exudate is found generally over the parietal and visceral peritoneum.

On examination of the intestinal loops there is found a semigangrenous process involving the jejunum from Treitz's ligament downward 18 to 24 inches. This segment of gut is of dark purplish red color and extremely friable. The visceral peritoneum of this involved gut strips off easily revealing edematous dark tissues beneath. The surface of the semi-gangrenous area exudes a serosanguinous fluid of much the same character as that found in the abdominal cavity. It is estimated that about 18 to 24 inches of the intestine were irreparably involved and that other segments of the upper jejunum were beginning to show some damage. The ileum is grossly normal. From the involved gut wall toward the root of the mesentery the veins are filled with antemortem clot and are found to be extensively thrombosed. As the veins are traced toward the root of the mesentery thrombus formation is less marked although many of the major vessels are completely occluded. All arteries seem free from antemortem clot. *Stomach:* contains large quantity of the dark colored thin fluid. *Liver:* slightly enlarged; is dark bluish, mottled with lighter areas of reddish yellow. Congested and rather soft. Scattered areas of yellowish discoloration similar to that seen on the exterior of the organ were found on section. These areas appear to be more intense around the central veins of the lobules. *Gall bladder:* chronically inflamed with rather extensive and firm adhesions around; mucosa thickened; no stones. *Spleen:* softer than normal, with some hemorrhagic congestion. *Kidneys:* congested, otherwise essentially negative. *Heart:* slight softening of cardiac musculature with apparently an antemortem clot in right auricle. Valves good condition. *Vascular system:* no arteriosclerosis.

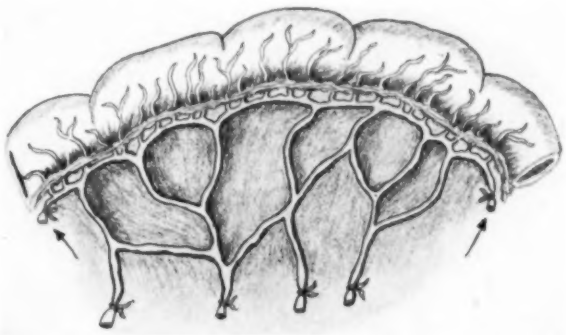

Fig. 1. Method used in procuring pure venous or arterial ligation, without direct lumen obstruction. For clearness, arterial and lymphatic trunks are not shown in diagram.

ANIMAL EXPERIMENTATION

In the animal experimentation in which we attempted to produce a picture simulating a venous thrombosis without direct

lumen obstruction we simply ligated the main primary venous radicals of the loop of intestine selected, together with the collaterals as illustrated in Figure 1. Upon tightening of the venous ligatures illustrated, an immediate bluish discoloration of the segment involved occurs and a stage of transient spasm of the muscular structure is rapidly followed by the soggy edematous condition of the gut wall which is seen grossly in the human condition of pure venous thrombosis. (See Figs. 3–4.) That actual gangrene does not usually occur in this condition of venous thrombosis may be demonstrated at any stage of the disease by stimulation of the muscular structure of the gut wall at the involved site. Naturally, therein lies the secret of the different clinical pictures of the arterial and venous conditions. It has been demonstrated time and again that destruction of arterial supply to even the smallest segment of gut which will produce gangrene, causes death very promptly. (See Table II.) We have invariably found in our dog series, that active muscular reaction occurred at any stage of pure venous thrombosis. Furthermore, bowel movements in all the dogs and in all the human cases continued until the time of death or until recovery. That the arterial system usually remains patent even until death or recovery, contradicts the opinion that venous thrombosis and stasis in the gut readily cause an arterial thrombosis. In Tables I and II one can readily see the marked difference in toxic symptoms in the arterial and venous types. Note the number of complete recoveries which occurred without surgery in the purely venous ligations.

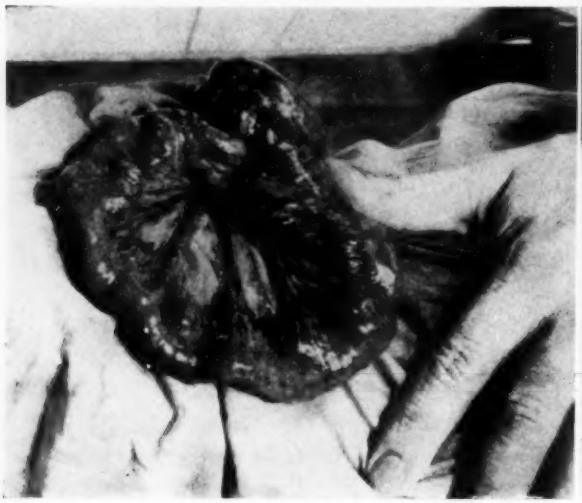

FIG. 2.

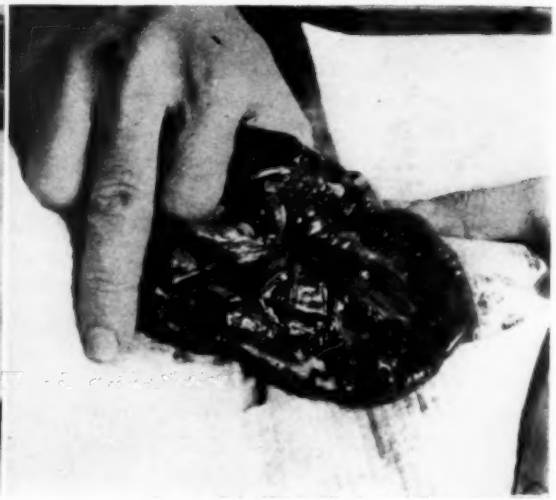

FIG. 3.

FIG. 2. Loop of normal lower ileum of dog in vivo. (Photographs by Haddow.)

FIG. 3. Same loop twenty-four hours after ligation of all veins of loop with their collaterals, after method illustrated in Figure 1. Note distended cord-like veins and very dark congested gut wall. (In vivo.)

FIG. 4. Same loop forty-eight hours after ligation. Gut wall in edematous and soggy but muscular reaction is still present. (In vivo.)

REMARKS

Since we started our work on venous mesenteric thrombosis, a case of venous thrombosis following repeated doses of

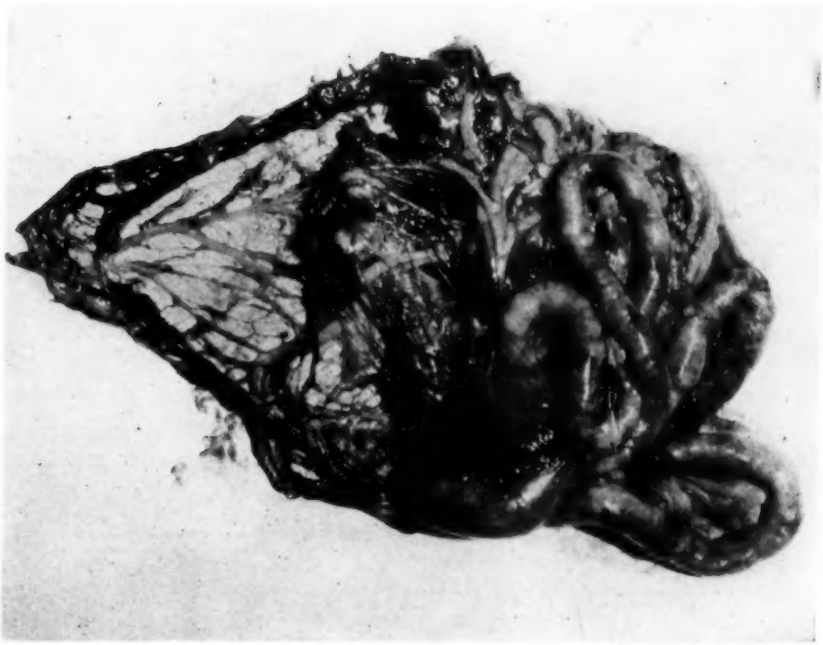

FIG. 5. Photo taken of same gut, dog having completely recovered clinically. Dog killed at third week. Baseball-sized area involved in mass of very firm adhesions. Omentum has firmly interwoven itself into involved site. Some collateral venous return seems to have been established through omentum. Note abnormally large omental veins leading from site. Bowels were moving normally and gut was completely viable.

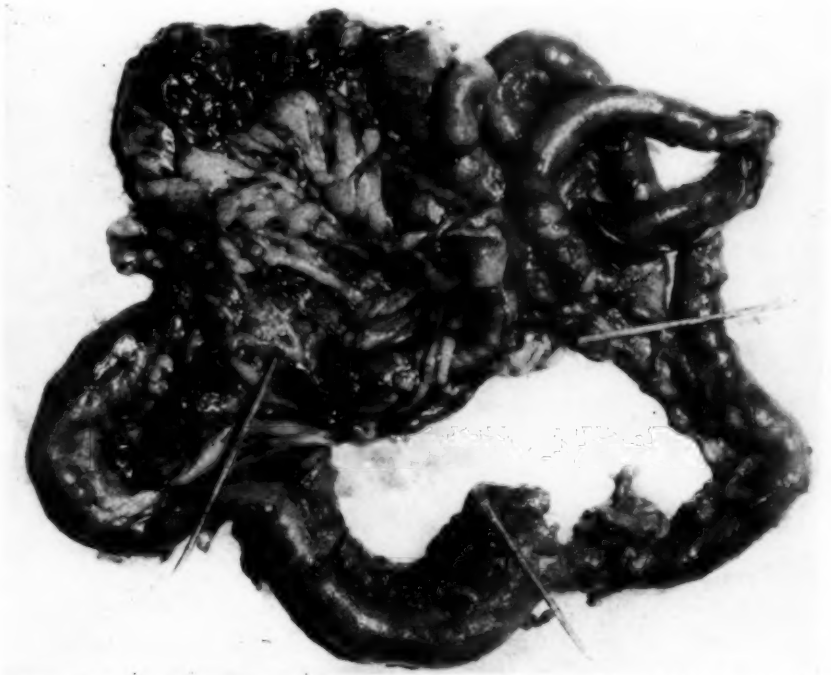

FIG. 6. Same gut loop which could not be disentangled without complete destruction of attachment of omentum and mesentery. Note length of area involved. All adhesions, though pronounced, were purely local. No general peritonitis developed in process of recovery.

phenolphthalein has been reported.[12] The similarity between this case and Wilson's case (Case II) which we have here recorded,

FIG. 7. Appearance of loop of lower ileum in vivo eighteen hours after arterial ligation. Note liquefying gangrenous condition of gut. Compare Tables I and II.

is striking. Note the possible relationship of the cathartics in both, together with the added insult of a heavy "Mexican" meal in Dr. Wilson's case. In two of the cases here reported, as well as the one just mentioned and another recently recorded,[5] note the several days over which the onset history extended.

One of us (J. K. D.) can readily recall an additional intestinal resection done three years ago in private practice in which venous thrombosis of an 18 inch segment of low ileum had undoubtedly been present two and one-half or three days before resection was done, with the patient ambulatory until the operation. She was, as a matter of fact, sent in the night before exploration, no particular emergency being recognized by us. The true character of the condition was not recognized at the operation, the gut being pronounced "gangrenous." No obstruction was present. Recovery was uneventful.

Case III, Dr. Johnson's, is reported here for two reasons. First, because though it is postoperative, it is a pure venous thrombosis; and secondly, because of the etiological interest attendant. We feel that Dr. Johnson has offered an explanation for the venous thrombosis which fits into our experiments and experience.

The possible low grade inflammation of a chronic enteritis associated with chronic cholecystitis may have been sufficient to have initiated a venous thrombosis.

We hope that this small series of human cases and animal experiments will serve as a stimulus to further work which may confirm and add to the value of our conclusions and opinions as they are outlined in this paper.

TABLE II
ARTERIAL LIGATION
(By method of Fig. 1)
(A venous or lymphatic radical, because of technical difficulties, occasionally included.)
Dogs

No.	Date	Area	Result
1	10–23–33	15 cm. low ileum	Died 20 hours. Moist gangrene.
2	12–30–33 11 A. M.	15 cm. upper ileum	Died 36 hours. Moist gangrene, progressing infarction to healthy bowel. No gross peritonitis.
3	10–30–33 11 A. M.	16 cm. low ileum	Died 27 hours. Moist gangrene—entire loop. No gross peritonitis.
4	11–20–34 12:30 P. M.	10 cm. high jejunum	Died 18 hours. Moist gangrene. Plastic adhesions, local.
5	12–3–34 2 P. M.	11 cm. high jejunum	Moribund 27 hours. Killed. Moist disintegrating gangrenous bowel. Plastic local adhesions.
6	12–3–34 2:30 P. M.	15 cm. low ileum	Moribund 27 hours. Killed. Moist disintegrating gangrenous bowel. Plastic local adhesions.

CONCLUSIONS

1. *Arterial and venous mesenteric thrombosis should be recognized and described as separate clinical entities.*

2. Arterial thrombosis is much more rapidly fatal than the venous type and must be operated on within a very few hours from the onset of gangrene, if recovery is to be anticipated.

3. The venous type is probably more prevalent than the arterial type; and is probably compatible with recovery without surgery in a certain percentage of cases.

4. The venous type is very amenable to surgery in the earlier stages.

5. We believe that it is possible in a goodly number of cases to recognize not only mesenteric thrombosis alone but that the type, as to venous or arterial, may be identified at times, preoperatively; and that the profession should discard the prevalent hopelessness which has been too often attendant upon the possibility of a correct preoperative diagnosis of mesenteric thrombosis.

6. Human cases and experimental work on dogs are herein reported in an effort to more clearly define the clinical picture of the venous type of mesenteric thrombosis.

7. We feel that a better understanding and closer inspection by the pathologist and surgeon of cases of venous mesenteric thrombosis will lead to the more frequent pathological diagnosis of hemorrhagic infarction of the gut rather than to the diagnosis of "gangrene."

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MATERNAL AND FETAL MORTALITY IN THE UNITED STATES*

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THE birth registration area in continental United States was established in 1915, at which time it included only ten states and the District of Columbia, representing 31.1 per cent of the population of our country. The area has been gradually extended since then by the addition of other states. In 1933, with the admission of Texas, it included for the first time the entire area for the continental United States. This was an important event as all comparative information regarding maternal and fetal deaths in this country is based on the statistics from the birth registration area. It seems appropriate then for us to consider the subject of maternal and fetal deaths in the United States at a meeting of the Obstetricians and Gynecologists of the last state to join the birth registration area.

During the past five years there has awakened in the minds of the laity in this country an unusual interest in maternal and fetal mortality. Such a public interest in this subject has also been witnessed in certain European countries and particularly in the British Isles. The reasons for this public awakening are fairly apparent. Obstetrics has steadily, although slowly, developed to be one of the major branches of medicine. However, it has become more and more evident to a small group of courageous ones in our profession that this development of the science and art of midwifery has not been attended by any marked improvement in maternal and fetal risks. In 1930, Comyns Berkeley of London stated that public opinion in England had at last been roused because of the fact that the maternal mortality and morbidity in the British Isles had not been lowered for the last twenty years, a statement which cannot be made about any other branch of medicine. In 1928, the Minister of Health of England appointed a Committee to study maternal mortality and morbidity in the British Isles. In 1932 this Committee made its final report, from which a great deal of important information may be obtained. Later I shall quote from this document.

In our country similar studies have been conducted by various societies during the past several years. The New York Academy of Medicine studied the problem in Greater New York City for the three years, 1930, 1931 and 1932, and published their findings and conclusions last year. Such investigations have been carried out in Philadelphia and other cities.

Concomitant with this deepened interest in this subject by our profession there have appeared, in the lay press and magazines, articles on almost every phase of maternal mortality. A great deal of this material has been erroneous and misleading, due undoubtedly to misinformation on the part of the writer. However, even if lay articles are often misleading the aroused interest of the lay public in the maternal mortality problem is focussing our attention on matters with which we should have concerned ourselves more diligently ere now. It is a commentary on our specialty that outside interest is needed to spur us on.

* From the Department of Obstetrics and Gynecology, Cornell University Medical College and New York Hospital. Read before the Texas Association of Obstetricians and Gynecologists, Galveston, Texas.

TABLE 1

DEPARTMENT OF COMMERCE
BUREAU OF THE CENSUS
DIVISION OF VITAL STATISTICS

DEATHS FROM PUERPERAL CAUSES WITH RATES PER 1,000 LIVE BIRTHS IN THE BIRTH REGISTRATION AREA IN CONTINENTAL UNITED STATES: 1915 TO 1932

Cause of Death	1915	1916	1917	1918	1919	1920	1921	1922	1923	1924	1925	1926	1927	1928	1929	1930	1931	1932
The puerperal state:																		
Number	4,719	5,091	8,938	12,496	10,127	12,058	11,688	11,792	11,922	12,672	12,158	12,168	13,837	15,461	15,084	14,836	13,964	13,120
Rate	6.07	6.21	6.61	9.16	7.37	7.99	6.81	6.64	6.65	6.56	6.47	6.55	6.47	6.92	6.95	6.73	6.60	6.32
Per cent of population of continental United States included in birth registration area	31.1	32.5	53.6	53.6	58.6	59.8	65.3	72.2	72.2	76.2	76.0	76.8	87.3	94.4	94.7			

Now why this interest, both professional and lay, in the matter of maternal mortality? Table 1 will best answer this question.

It is clear from this table that no real improvement in the mortality figures has taken place during the past twenty years. It is also well to remember that deaths due to "the Puerperal State" as classified by the United States Bureau of the Census exclude such deaths as those due to one of the more serious acute infectious diseases. In a study of maternal deaths in 15 states by the Children's Bureau under the Department of Labor, in which the deaths were classified by the United States Bureau of the Census, we find the definition of "the Puerperal State" as follows:

1. Accidents of pregnancy. This includes (a) abortion, (b) ectopic gestation, and (c) others under this title. ("Abortion" will be referred to throughout this report as "abortion or premature labor." The word "abortion" does not have the same meaning as it does in the international classification, but is defined as the termination of a uterine pregnancy before the period of viability, i.e., in the first two trimesters.)

2. Puerperal hemorrhage, which includes (a) placenta previa, (b) others under this title: postpartum hemorrhage, accidental hemorrhage, etc.

3. Other accidents of labor.
   (a) Caesarean section.
   (b) Other surgical operations and instrumental delivery.
   (c) Others under this title.

4. Puerperal septicemia.

5. Puerperal phlegmasia alba dolens, embolus, sudden death.

6. Puerperal albuminuria and convulsions. This title also includes pyelitis, nephritis, tetanus and uremia.

7. Following childbirth (not otherwise defined).

8. Puerperal diseases of the breast.

In further elaboration of the definition we read:

When more than one puerperal cause appears on a death certificate, the death is assigned to one of them in accordance with definite rules

published in the Manual of Joint Causes of Death, which the Children's Bureau has followed literally in all cases. It is well to realize what the general rules of the classification are. If one of the more serious acute infectious diseases, such as typhoid fever, smallpox, diphtheria, or if cancer or syphilis, or if an external cause such as an accident or homicide, appears on a woman's death certificate with a puerperal cause, her death is assigned to that cause and not to the puerperal cause. (Influenza, however, takes precedence over no puerperal cause except "other accidents of pregnancy," "following childbirth (not otherwise defined)," and "puerperal diseases of the breast.") Puerperal septicemia takes precedence over all puerperal and non-puerperal causes except the ones mentioned. Tuberculosis in most forms takes precedence over all puerperal causes except puerperal septicemia. Other serious chronic diseases, such as cardiac valvular disease and chronic nephritis, take precedence over all puerperal causes except the most severe complications of childbirth. The term "pregnancy" on a death certificate causes a death to be classified as puerperal only when it appears alone, or with a term denoting a mild disorder or with a cause implying a complication of pregnancy.

It would appear then that the maternal mortality is even worse than is indicated in the foregoing table. I should like to see all countries and clinics adopt a uniform method of reporting maternal mortality or deaths due to "the puerperal state." This death rate should express the percentage of women, with both nonviable and viable babies, dying during pregnancy, labor and the puerperium, *without* correction for such complications as heart disease, cerebral accident, anemia, thrombosis, embolism, pulmonary edema, pneumonia, influenza, tuberculosis, diabetes, chorea, epilepsy, appendicitis, criminal abortion or any other complication of pregnancy. Until all maternal deaths are reported in this manner, statistical comparisons are futile, for the reason that all other methods permit of correction, and unfortunately all writers, investigators and countries do not correct in the same manner. Therein lies the trouble.

TABLE II

DEATHS FROM PUERPERAL CAUSES WITH RATES PER 1000 LIVE BIRTHS IN THE BIRTH REGISTRATION AREA IN CONTINENTAL UNITED STATES: 1930

Causes	1930	1931	1932
Abortion with septic conditions:			
Number	1961	2049	2026
Rate	.88	.96	.97
Abortion without mention of septic condition (to include hemorrhage):			
Number	671	653	706
Rate	.30	.30	.34
Ectopic gestation:			
Number	595	588	562
Rate	.26	.27	.27
Other accidents of pregnancy (not to include hemorrhage):			
Number	169	88	84
Rate	.07	.04	.04
Puerperal hemorrhage:			
Number	1523	1442	1377
Rate	.69	.68	.66
Puerperal septicemia (not specified as due to abortion):			
Number	3321	3149	2734
Rate	1.50	1.49	1.31
Puerperal septicemia and pyemia:			
Number	3303	3137	2721
Rate	1.49	1.48	1.31
Puerperal phlegmasia alba dolens, embolus, sudden death (not spec. as septic):			
Number	702	630	626
Rate	.31	.29	.30
Puerperal tetanus:			
Number	18	12	13
Rate	*	*	*
Puerperal albuminuria and eclampsia:			
Number	3589	3027	2659
Rate	1.62	1.43	1.28
Other toxemias of pregnancy:			
Number	493	529	489
Rate	.22	.25	.23
Other accidents of childbirth:			
Number	1767	1755	1807
Rate	.80	.83	.87
Other and unspecified conditions of the puerperal state:			
Number	45	54	50
Rate	.02	.02	.02

* Less than one-tenth of 1 per 1000 live births.

Furthermore, you will notice that in my definition of maternal mortality as already stated, I said "the percentage of women . . . dying," and not "deaths per 1000 live births." I am aware of the reason usually given for computing maternal mortality on a basis of live births, namely that it expresses the sacrifice in mothers to produce so many live births. This method leads to numerous errors and affords many ways of correcting the death rate. The higher the fetal mortality, the lower the number of live births, a state of affairs affecting the maternal death rate. In other words, the maternal death rate depends not only on the actual number of women who died as a result of childbirth but also on the number of babies born alive. This, to my mind, is a most illogical method of recording our results as they relate to the mother. I believe we should be able to state quite clearly that out of a given number of women who became pregnant the death rate was thus and so; in other words, so many puerperal women died per 100, or 1000, women who became pregnant. I advocate very strongly that all maternal mortality reports, either by individuals, societies, cities, states or countries, be based on the total uncorrected maternal deaths, irrespective of the duration of pregnancy or the result to the offspring.

Let us consider the main causes of maternal mortality as shown by the figures of the United States Bureau of the Census, bearing in mind the foregoing comments on "the puerperal state" and on "deaths per 1000 live births." In Table II will be seen the statistics for the last three years ending 1932. These show very clearly that puerperal infection is the main cause of death with a rate of almost 3 per 1000, or nearly 50 per cent of all deaths. The toxemias appear next with a rate of 1.5 to 2 per 1000. Puerperal hemorrhage is the third largest contributing factor, accounting for 0.7 per 1000. These three: infection, toxemias and hemorrhage, constitute then the cause of death in almost 85 per cent of all fatal cases.

I should like to present to you now the figures on maternal mortality in the Woman's Clinic, or Lying-In Hospital of the New York Hospital. These are shown in Table III.

I do not present these figures in order to impress you with our results, but rather to bring out the reasons, as I see them, for such figures. Our morbidity incidence is low because of rigid aseptic technique and an isolation floor for all infected or potentially infected patients. This isolation floor is completely equipped with nurseries, kitchens, sterilizing room, operating and delivery room, and is subdivided into sections, one for observation and the milder forms of infection such as pyelitis, gonorrhea and breast infections and one for puerperal infection.

It should be noted that the morbidity rates as shown in Table III are uncorrected and give the results in a maternity hospital where both registered and unregistered, as well as emergency patients are admitted and treated. These figures, therefore, permit of comparison with any other series of cases reported on an uncorrected statistical basis.

Regarding morbidity, I recently stated:

> It is evident that a bidaily temperature reading often gives very false and misleading information, especially if such temperatures are taken early in the morning and at two o'clock in the afternoon. The four and eight o'clock temperatures are more significant than that at two o'clock. On such a bidaily basis of recording temperatures, many patients with an uterine infection, and even with a hectic temperature course may appear to be afebrile. The important factor in detecting a puerperal infection is that throughout the patient's stay in the hospital her temperature be faithfully recorded at 6 and 10 A.M., 2, 6, and 10 P.M. and 4 A.M. The last temperature, 4 A.M., may be omitted if the patient is asleep. Our standard of morbidity in the Woman's Clinic of the New York Hospital is as follows: "The Puerperium is considered febrile if there is a rise in temperature to 38°C. (100.4°F.) occurring once during each of two twenty-four hour periods

following delivery, or remaining elevated longer than twenty-four hours, excluding the first twenty-four hours after delivery, temperatures on all patients being recorded every four hours throughout the patient's stay in the hospital."

Other factors besides a rigid admission system and adequate isolation, which lower the incidence of infection, are the use of a face mask by all attending a patient in labor and at delivery, surgical asepsis on the part of the obstetrician, the proper performance of rectal examinations and elimination, as far as possible, of vaginal examinations during labor, a low incidence of operative interference, a house staff adequately trained in obstetrics, adequate supervision by the attending staff and careful nursing care during labor and the puerperium, especially in so far as perineal care is concerned. I may say that our house staff is on a five year resident system.

The prevention, control and treatment of the toxemias is too large a subject for me to discuss at this time. Suffice it to say that antenatal care, properly instituted and conducted, the conservative treatment of pre-eclampsia and eclampsia, the early recognition and treatment, radical if necessary, of nephritis as well as a careful follow-up system for a period of six months to one year of all cases are the important factors in any attempt to lower the deaths from the toxemias.

As to the third important cause of maternal deaths, hemorrhage, I may say that our treatment has changed most radically during the past ten years. Today a patient bleeding, however little, during the last trimester of pregnancy, is brought into the hospital and the *first* step in the treatment is blood grouping and matching. This is done before any other step in the procedure, unless some type of obstetrical

TABLE III

MATERNAL MORBIDITY AND MORTALITY IN THE WOMAN'S CLINIC, NEW YORK HOSPITAL FOR THE PERIOD SEPT. 1, 1932 TO JUNE 30, 1934

A. *Morbidity (Indoor Service)*

	Premature		Full Term		Total	Per Cent Total	Per Cent Morbidity
	Spont.	Oper.	Spont.	Oper.			
Puerperium: Normal	75	36	2718	630	3459	74.244	
One day fever	11	4	406	153	574	12.320	
Febrile, Puerperal infection	12	9	228	176	425	9.122	13.436
Mastitis	0	0	37	10	47	1.009	
Pyelitis	2	2	22	5	31	0.665	
Intercurrent disease	1	1	14	3	19	0.408	
Other causes	2	6	57	39	104	2.232	
	103	58	3482	1016	4659	100.000	
	161		4498				

B. *Mortality*

Total indoor full term + premature deliveries	4659
Total deaths in indoor service	7
Incidence = 0.15 per cent or 1.5 per 1000	
Total obstetrical patients treated in indoor & outdoor service	7869
Total maternal deaths	14
Incidence = 0.177 per cent or an uncorrected maternal mortality of 1.77 per 1000	

interference is imperative. Many of these patients stop bleeding and it is our practice in such instances to refrain from vaginal examinations for four or five days, and these are carried out only when definitely indicated. The prime importance of adequate preparedness at all moments in the matter of blood transfusion for the case of puerperal hemorrhage has become more clear to us and it has displaced immediate obstetric interference from first place in the routine procedure of treatment.

Time will not allow me to enter into a detailed discussion of the other various causes of maternal mortality, but later I shall summarize my views as to the factors in general which do and will lower our appalling maternal mortality.

The confusion we have seen in the reporting of maternal mortality is surpassed only by that observed in the statistics on fetal mortality. There is no single definition of fetal mortality in general use and the application of the terms stillborn and deadborn vary widely in the law and practices of the different states. I have recently advocated that fetal deaths be reported in the form of total uncorrected infantile mortality rate, including all stillbirths in babies of 1500 gm. or over, as well as neonatal deaths occurring during the first fourteen days of life. This is the only basis on which intelligent comparisons can be made. A corrected fetal mortality rate is as misleading as a corrected maternal mortality rate.

It will be seen from this definition that no corrections are made for premature babies nor for neonatal deaths, occurring during the first two weeks of life. This definition obviates any argument as to what constitutes viability and brings to the light of day the bad results, occurring one or more days after delivery, and following birth injuries incident to such obstetrics as unskillful forceps application, unnecessary haste in breech extraction unwarranted and untimely version and extraction, as well as all the other methods of meddlesome and unskilled midwifery.

The figures of the Bureau of the Census of the United States on fetal mortality give merely the number of stillbirths, and so do not tell us the whole story. But from those figures alone it would seem that our results may stand great improvement. In 1933 there were 2,064,944 births in the continental United States with 76,837 stillbirths, a rate of 3.7 per 100 live births. We do not know the number of premature and neonatal deaths. I would estimate that the total uncorrected infantile mortality in the United States is in the neighborhood of 6 per cent. I base this estimate on the fact that in a large maternity service we have found that the neonatal deaths constitute approximately one-third of the total infantile mortality. For example, in our clinic for the year 1933 the gross infantile mortality was 145 deaths, in 3752 infants, or 3.86 per cent, and of these 145 deaths, 47 occurred during the first two weeks following delivery.

It is often stated that the maternal and fetal mortality in other countries, especially the Scandinavian, are far lower than in the United States. Such comparisons are dangerous and misleading. I quote for you from the Final Report of the British Committee on Maternal Mortality which made an exhaustive study of the Scandinavian countries. Their report states:

> In Denmark and Sweden the official maternal death rates for the five years 1925–29 average 2.74 and 3.12 respectively, as compared with 4.21 for England and Wales. Enquiry showed, however, that certain causes of death, which in the English returns are classed to childbirth, were habitually excluded from this category in Denmark and also that in both these countries the method of classification where more than one cause of death appears on the certification diminishes the number of cases which would in England have been ascribed to maternal deaths. The investigators arrived at the opinion that in consequence of these discrepancies the official figures as they stand do not give a true idea of the relative maternal mortality, and that if the rates in these countries were computed upon the English basis they would more closely approach that of England and Wales. In all the countries there has been a definite rise in the recorded

maternal mortality rate in recent years. Different reasons for this were given in each country, but it would seem that in all the greater care now exercised in death certification has been an important factor in bringing about this result.

Furthermore, if we consider that for the year 1929 the total maternal mortality for England, according to this report was 5.82 deaths per 1000 live births, while in the United States for the same year the figure was 6.9 for colored and white, and 6.3 for its white population, it would appear that the standard of obstetric practice is about the same in each of these countries, and leaves much to be desired.

To my mind maternal mortality in the United States can be materially reduced by allowing more time for the teaching of obstetrics in our medical schools, by postgraduate teaching in obstetrics, by better hospital training in obstetrics for those who deliver women in their practice and by providing adequate welfare clinics and hospital facilities for maternity patients. These facilities should include, as so well set forth by the American College of Surgeons, the following:

a. Segregation of obstetric patients from all others in the institution.

b. Special facilities available for immediate segregation and isolation of all cases of infection, temperature or other conditions inimical to the safety and welfare of patients within the department.

c. Adequately trained personel, the entire nursing staff to be chosen specially for work in this department and not permitted to attend other cases during time on obstetric service.

d. Readily available, adequate laboratory and special-treatment facilities under competent supervision.

e. Accurate and complete clinical records on all obstetric patients.

f. Frequent consultations encouraged on obstetric service, a consultation made obligatory in all cases where major operative procedure may be indicated.

g. Thorough analysis and review of the clinical work of the department each month by the medical staff with particular considerations to deaths, infections, complications, or such conditions as are not conducive to the best end results.

h. Adequate theoretical instruction and practical experience for student nurses in prenatal, parturient, and postpartum care of the patient, as well as the care of the newborn.

Finally I feel very strongly that the general public should be taught that it is imperative for *every* woman to have adequate medical supervision and care during pregnancy, labor and the puerperium. This supervision and care must begin early in pregnancy, must be continuous throughout the postpartum period and must be in the hands of a physician, not a midwife, properly taught as an undergraduate and adequately trained in a first class hospital in the art and science of obstetrics.

Regarding certain other specific factors relating to maternal mortality, such as anesthesia and analgesia, midwives and home deliveries, I can do no better than to quote from the Report of a Committee of the New York Obstetrical Society appointed this year to review the Maternal Mortality Report of the New York Academy of Medicine. As I was a member of this committee, my views are in complete accord with the following quotations from its report. These are:

Midwives:

Your Committee believes that while there is need for better training and supervision of the present licensed midwives, that there is no need for training or licensing of additional midwives. The record shows that during the past twenty years in the city of New York there has been a steady decline in the practice of midwives—from 50 per cent to 8.5 per cent at the present time. In order to accomplish the better training and supervision of the existing midwives, we recommend that the New York Obstetrical Society offer its services to the Commissioner of Health of the City of New York.

Your Committee believes that the practice of Obstetrics will never be elevated to the position it rightly deserves as long as the midwife is permitted to practice. The profession, as well as the majority of the laity, is perfectly cognizant of the limitations of the midwife and the teachers and leaders of obstetrics should, by now, appreciate the inadequacy of

any system which introduces incompetency in competition with scientific knowledge.

Hospital and Home Deliveries:

Your Committee recommends that the New York Obstetrical Society should countenance home deliveries only when under ideal conditions as to competent medical attendance or supervision, equipment and assistance.

Operations:

We all know that operative deliveries, with good indications in skilled hands, are necessary. They are merciful and life-saving and they constitute one of the great advances of modern obstetrics.

Anesthesia and Analgesia:

Your committee believes that the use of anesthetics and analgesics when properly selected and properly administered are valuable and indispensable and should be encouraged. They are not only humane but tend to prevent unnecessary and too early interference with the natural progress of labor and *per se* do not add to the maternal or fetal death rate, nor to operative interference.

Education:

Your Committee is unanimously of the opinion that the New York Obstetrical Society should go on record in the medical and lay press as to its attitude regarding education and training in obstetrics and gynecology. This seems necessary in order that the Society may use its full influence in an endeavor to accomplish the following: Hospital training in obstetrics for those who plan to practice obstetrics must be adequate. An obstetrical interneship should not be less than one year and a general rotating interneship in which obstetrics is included, should be at least two years in order to allow a minimum of six months for obstetrics.

All university and other qualified hospitals should utilize their facilities, wherever possible, to offer postgraduate courses in obstetrics and gynecology.

Only through these three factors, adequate undergraduate teaching, thorough hospital training and postgraduate courses, will the obstetrical standards of the work in all our hospitals be elevated to the required level of efficiency and safety.

Furthermore the lay public, through the various public health agencies, including the Department of Health, should be informed of the requirements of a physician doing obstetrics; the standards of hospitals taking obstetric cases; and finally what the patient herself should know about pregnancy, labor and the puerperium.

Cooperation with Medical Organizations and Publicity to the Medical Profession:

Your Committee recommends that the New York Obstetrical Society appoint a Committee to cooperate with the Department of Health, the Department of Hospitals, and other agencies, in order to accomplish the above recommendations regarding midwife practice, undergraduate and postgraduate education, regulation or registration of physicians practicing obstetrics, supervision of all hospitals doing obstetrics which do not conform to the minimum standards of the American College of Surgeons and to evolve a plan for a Board of Regional Consultants in obstetrics and gynecology. We further recommend that, with respect to our report, such a Committee be empowered to consider and act in the matter of publicity to the medical profession, as well as to the lay public.

Fetal Mortality:

Your Committee recommends that in any further study of Maternal Mortality, fetal mortality should be included as giving more complete and accurate analysis of our results in childbirth.

I have endeavored to bring before you the appalling figures, seen in every state in the Union, on maternal and fetal mortality and have further attempted to indicate lines along which we may hope to elevate the standard of obstetrics and gynecology in our country, which will automatically carry with it marked improvement in these frightful mortality figures. It rests with you and me, as obstetricians and gynecologists, to vindicate ourselves in the eyes of the general public, as well as our own profession. We must accomplish in our specialty what is being done in other branches of medicine, we must prolong the span of woman's life, we must make childbirth safe and without undue risk to mother and child and we must see that womankind suffers not from the end results of inadequate obstetrics. This is our task and the responsibility may not be shifted.

SUPPORT OF THE PELVIC VISCERA

AND THE MECHANISM OF PROLAPSE

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INTRODUCTION

IN a previous paper,[1] after reviewing briefly the lack of value of the other ligaments as supporting structures to the uterus, it was shown by histologic section on autopsy and operative material, that there was no ligamentous tissue in the base of the broad ligament around or near the uterine vessels and, extending from the uterus out to the lateral pelvic wall, as described in some texts under the name of the Mackendrodt, the cardinal, or the utero pelvic ligaments.

It was shown that the uterus cannot depend for support on any structures to be found in the base of the broad ligament, any more than it can on any other and all the tissues to which it is attached. Therefore the explanation of the development of prolapse of the uterus can no longer include a consideration of these so-called ligaments. There remain only two other possible causative factors: (1) pressure from above, both intra-abdominal and intrapelvic, and (2) lack of support from below. If attention is directed only to the descent of the uterus it is very likely that, as has happened in the past, much of importance will be missed because the descent of the uterus is merely one symptom of an underlying lesion. Attention should be focused on the fact that there is a bony outlet at the bottom of the pelvis and that unless some adequate means for closing it is provided, the visceral contents of the pelvic and abdominal cavities will be extruded by the combined forces of gravity and intra-abdominal pressure.

The bony outlet is completely closed by the levator ani muscle excepting in its anterior median portion. Here there is a deficiency for the passage of the urethra, vagina and rectum, called the pelvic floor aperture. It is this aperture caused by a deficiency in musculature about which the whole problem of prolapse centers. If there were no such deficiency, there never could be any prolapse. Manifestly then, knowledge of how that aperture is normally kept closed will furnish an important aid in the elucidation of the problem of the prolapse mechanism.

The continued teaching that the pelvic aperture is normally closed by fascial structures, the integrity of which prevents prolapse, betrays an utter disregard of accurate anatomical, physical and pathological data which have been separately accumulated over a long period of years. With logical application of the data a solution can be evolved which is in accord with all known facts, is not theoretical, or supposititious, and offers in turn, a sound explanation for the mechanism by means of which prolapse is prevented normally.

CLOSURE OF BONY OUTLET

The bony pelvic outlet is diamond shaped. The contents of the pelvis are prevented from being extruded through the outlet by a muscular hammock consisting of the levator ani and the coccygei muscles which completely closes the bony outlet except for a slight deficiency anteriorly in the median line.

The levator ani arises from the pubis about ½ inch from the middle of the symphysis, from the fascia covering the obturator internus muscles (the so-called "white line") and from the ischial spines. These fibers pass backward and inward obliquely, the anterior running more directly backward and the posterior ones more nearly inward. They are inserted into the anococcygeal raphe or ligament and into the coccyx and terminal part of the sacrum. The anterior free margins of the levator ani form the lateral boundary of the pelvic floor aperture, the deficiency in the pelvic floor. The pubis forms the anterior boundary and the junction of both levators form the posterior boundary of the deficiency. The posterior margins of the levator ani overlap the anterior free margins of the coccygeus while the posterior free margins of the coccygeus overlap the small sacrosciatic ligaments. Excepting for the deficiency anteriorly in this musculature the opening of the bony pelvic outlet is perfectly closed by these muscles. Through this deficiency the urethera, the vagina and the rectum gain an exit to the surface of the body. This aperture situated medially and anteriorly just behind the pubic arch is obviously a necessity.

If this pelvic floor aperture remained open constantly the pelvic viscera would be in danger of extrusion not only because of the force of gravity but also because of the continuous variation in intra-abdominal and intrapelvic pressure (caused by the contractions of the diaphragm and the abdominal wall by coughing, sneezing, straining, etc.) which would constantly compress and force the pelvic viscera out. There must exist some mechanism which would be able to keep this pelvic floor aperture adequately closed to counteract the extruding force of this pressure and yet provide for the occasional passage through the aperture of the contents of the three canals which traverse it. It must therefore be both resistant and elastic. Consequently, it is inconceivable that such a closure-mechanism could consist of fascial structures which are inherently dense and unyielding or else have no supportive value at all. Certainly no fascial structures whether dense and firmly resisting or loose and yielding could be adaptable to the requirements placed upon this aperture of both closure and relaxation for the passage of visceral contents.

It has been the opinion of many that the pelvic fascia which forms a continuous sheet varying in density in certain positions, is the chief support of the pelvic viscera against ordinary conditions of pressure. Studies indicate quite definitely that the development of the fascial sheath of muscles depends on the functional activity of the muscle. The analogy which has often been drawn between the value of the fascial support of the abdominal wall and the fascial support of the pelvis is not based upon proper premise. The fascial support of the abdominal wall is not a fascial investment of muscle; it is an *aponeurosis* of origin or insertion of muscles. The individual muscles have their own fascial coverings, but the aponeurosis of the external oblique muscles, for example, is a very dense, highly resistant broad tendinous fibrous tissue sheet which forms the insertion of the muscle. The fascial sheath of the muscles of the abdominal wall is very much more delicate than any aponeurotic continuation of any of the muscles of the abdominal wall. Such aponeurotic membrane cannot be compared to the very much more delicate, thinned-out, and less resistant fascial sheath of the pelvic floor musculature as a restraining factor in the prevention of prolapse. Goff[2] demonstrated very clearly that in the rectovaginal and the vesicovaginal septum, the only tissue which can be found which might be called a fascial structure is a loose areolar connective tissue. This cannot be dissected as an individual layer. Thus there is no fascia other than this loose areolar tissue available for use in the surgical correction of

either cystocele or rectocele. This has been corroborated by me.[1]

Instead of fascia, however, there is a musculature which, with the exception of the anterior pubic arch, completely circles the pelvic floor aperture and the three canals which pass through it. This musculature, when intact, is capable of increased contraction and great relaxation and forms a very efficient closure-mechanism capable not only of preventing the extrusion of abdominal and pelvic viscera, but also of permitting the proper passage of visceral contents from the pelvis to the exterior. Let us now carefully consider how it effects the closure of the pelvic floor aperture.

This musculature, in the region of the pelvic floor aperture consists of two distinct parts, the pubococcygeus and the puborectalis. The former, is more or less in the same plane as, and continuous posteriorly with, the remainder of the pelvic floor musculature, the ileococcygeus and the coccygeus. The latter, the puborectalis, is placed just below this in a different plane entirely. Both arise from the pubic bone, their innermost border reaching so near the symphysis that the distance from the inner edge of the muscle bands of one side to that of those on the other is 1 inch. The upper, the pubococcygeus, passes backward, inward in a gentle curve and slightly upward to be inserted into the anococcygeal raphe and coccyx forming with its fellow of the opposite side a V-shaped muscle. The apex of this V is at the coccyx while the diverging arms form with the pubis the upper part of the pelvic floor aperture. The lower, the puborectalis, passes backward by the side of the vagina and rectum but instead of being attached to the coccyx or anococcygeal raphe turns around the anorectal junction and becomes continuous with the corresponding muscle layer of the other side. The two muscle arms together form a U-shaped muscle, the *Posterior Commissure of Which Is Not Tendinous or Fibrous but Continuous Striated Muscle Lying Free in Loose Fatty Connective Tissue.* At this point the puborectalis lies above the level of the superficial perineal muscle but below that of the main part of the pelvic floor musculature. Its muscle arms extend forward to just above the anterior border of the pubic arch and form with that part of the arch between the points of origin the lower and more important part of the pelvic floor aperture. It must be remembered that unlike the pubococcygeal muscle the puborectalis has no posterior attachment either to the anococcygeal raphe, the coccyx or to the sacrum. (Figs. 1, 2.)

When muscles contract they shorten in the long diameter and thicken transversely. If they have bony attachments at both ends, resistance to the shortening results in increased tension of fibers. In the case of the pubococcygeus, a bilateral musculature having bony attachments at both ends is so arranged because of its gentle curve that between the two symmetrical parts there exists an almost elliptical space. Contraction of such musculature with its consequent thickening gives rise to a diminution in the width of the space. Thus, the pelvic floor aperture is diminished in its transverse diameter when that muscle contracts. This contraction results in a bilateral occlusion of the visceral canals placed in the median line between the two symmetrical arms of the musculature. It has been definitely shown that the rectum is constricted in this way.[3]

The result of the contraction of the puborectalis is entirely different. It was emphasized previously that it has a bony attachment anteriorly but no tendinous or bony connections posteriorly. When it contracts, there is no bony resistance to shortening in the longitudinal diameter and the posterior commissure of this muscle is pulled forward towards the pubic arch. In its forward advance it pulls the rectum forward at its flexure and in so doing pushes the vagina firmly against the pubic arch. In this way it acts as a very efficient sphincter mechanism for closing

the pelvic floor aperture, thus compressing and closing the two visceral canals, the vagina and rectum. Budin[4] has shown that

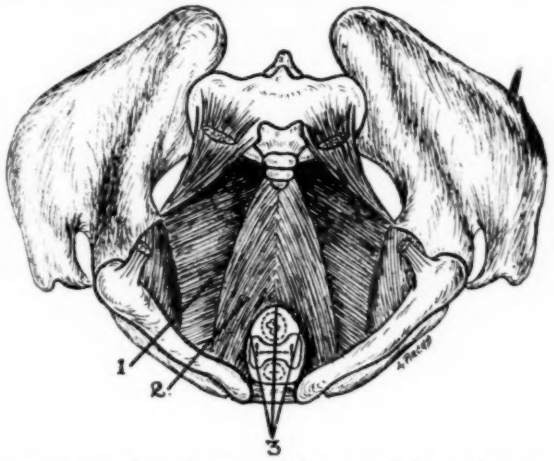

Fig. 1. Muscular closure of bony pelvic outlet. 1, Lateral portion of levator ani. 2, Pubococcygeus portion of levator ani. 3, Pelvic floor aperture where a deficiency in musculature exists.

a cylinder of wax inserted into the vagina during voluntary muscular relaxation receives a very marked circular impression upon the forceful voluntary contractions of the muscles of the pelvic floor. Although the impression is most marked in the antero-posterior direction, particularly on the side toward the pubic arch, the lateral indentations are quite definite. The marked indentations of the wax model are evidence not only of the sphincteric closure of the vagina but also and more important they are further evidence of the value of the contraction of the puborectalis muscle as a means for closure of the pelvic floor aperture by the forward advance of the posterior commissure of the muscle. Even when the external sphincter has been completely lacerated the retention of solid feces within the rectum is made possible by the contraction of the puborectalis portion of the levator ani muscle. Its sphincteric action in closing the vagina plays an important part during coitus, sometimes causing imprisonment of the penis within the vagina. Such cases have been described by Hilderbrandt,[5] Budin[4] and others. Dickinson[6] has noted the particularly good development of this muscle in erotic women.

If this aperture were very large its closure would be very difficult but its anteroposterior diameter measured from

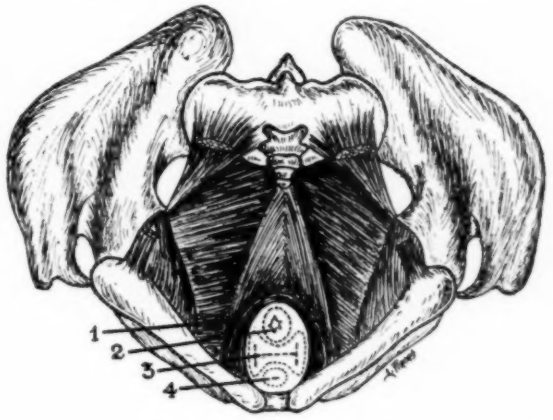

Fig. 2. Muscular closure of pelvic floor. 1, Puborectalis muscle at a lower plane than pubococcygeus and having no bony attachment posteriorly. 2, Rectum. 3, Vagina. 4, Urethra.

the midpoint of the summit of the pubic arch at the symphysis to the posterior wall of the anorectal junction is about 1½ inches. At first this seems grossly underestimated, but careful examination reveals it to be an accurate measurement. It must be remembered that this aperture is not a patent foramen with no contents. On the contrary it is completely filled by the tissues forming the urethra, vagina and rectum. That these structures do not fall out is due to the fact that circumferentially they are enclosed by or adherent to the muscle bounding the aperture. Henle[7] has shown that the non-striated muscle of the rectum and vagina interweave with the striated fibres of the puborectalis. Furthermore, these three visceral canals are adherent to each other and their mechanical presence reduces the size of the pelvic floor aperture so that it really exists only potentially during the resting stage.

SUPPORT OF THE PELVIC VISCERA

There is no free space in the pelvis. The entire pelvis is occupied by viscera. Each viscus possesses volume and although it may be deformable it is incompressable. The thoracic diaphragm and the anterior abdominal muscular walls are continually

in apposition with the viscera contained within the abdominal cavity and constantly compress them as a result of their muscular tonus. This produces a pressure amongst them which is transmitted to the pelvic viscera and causes the latter to press against each other and in addition upon the walls and floor of the pelvic cavity. Since the bony pelvis is unyielding, the continuous changes in volume of some of the pelvic viscera must be made at the expense of other organs.

Concomitant with breathing, coughing, sneezing and straining movements, changes constantly occur in intra-abdominal and the intrapelvic pressure to which the pelvic viscera are subjected. This pressure, exerted upon the viscera in all directions but especially so in a downward direction wherein it is increased by the force of gravity, tends to cause their protrusion from the pelvis. This forms one component of a delicately balanced mechanism. The intact musculature of the pelvic floor, operating as already described, is the only resistant power so placed as to be capable of preventing such extrusion and it constitutes the second component. The innervation of the musculature allows for rapid reflex accommodation by contraction and relaxation to changes in intra-abdominal and intrapelvic pressure.

The pubococcygeus muscle is present in four-legged animals. There is no puborectalis muscle in such animals. In them the rectum is flattened laterally by the two pubococcygeus muscles, the free borders of which stretch between the symphysis pubis and the mid-line of the root of the tail, and in them the rectum runs a straight course. As a result of the assumption of the erect posture the puborectalis muscle was evolved, to provide a compensatory mechanism to control the increased intra-abdominal pressure which, unobstructed, might bring about an extrusion of the pelvic viscera.

Examination of the puborectalis in the dissecting room where it is seen in a distinctly atrophic state is partially responsible for the old idea that it can have very little value as a restraining influence in the pelvic floor diaphragm. To properly appreciate that it exists, lives and really functions, however, it must be studied in the living subject. This is best done in corpulent or pregnant women in whom there has been a rise in intra-abdominal and intrapelvic pressure which is followed by an adaptive hypertrophy of the muscles. In a well-developed primagravida, this muscle can easily be palpated as a tense mass of tissue constricting the finger against the pubic arch and it can be traced alongside the vagina, from its pubic origin backward. On simultaneous rectal examination with the other hand it can be perceived to come from beside the vagina backward by the side of the rectum and then inwards behind and around the anorectal junction posteriorly where it becomes continuous with a similar mass of tissue on the other side. This in turn can be followed forward to the pubic bone of the other side. It lies at a higher level than the external sphincter of the anus. Straining movements on the part of the individual during examination will cause a marked contraction of the muscle. This will force the fingers against the pubic arch. It is readily distinguishable from the superficial muscles which are inserted into the perineal body. If it be picked up between the finger and thumb, it can be felt to harden, that is, to contract on coughing and straining. This contraction which is felt is live muscle function and not passive fascial action to which so much importance has previously been laid.

Thus the conception is established that the pelvic floor aperture is closed by a living muscular mechanism. The association between increased abdominal pressure and contraction of the circular musculature surrounding the pelvic floor aperture can be voluntarily interrupted as for example during defecation but unless conscious dissociation is created the muscle contracts reflexly as a protective mechanism against extrusion of contents of the

pelvis or of the canals running through the pelvic floor aperture.

In a standing position, the pelvic floor aperture is in the most dependent portion of the pelvic cavity and might seem to be most disadvantageously placed. Such position would invite the assumption that in prolapse it is the weight of the individual organ plus the effect of gravity which causes the extrusion. This, however, is not true because the patient suffering with prolapse, after reposition of the organs, even placed in the lateral position, can voluntarily, by bearing down, extrude the pelvic organs from the pelvic floor aperture.

Considering the position of the pelvic floor aperture in relation to the transmission of increases of intra-abdominal pressure to the pelvic cavity, it would at once seem that the aperture is placed in the most advantageous position against prolapse. A sagittal section of the body shows the outline of the abdominal cavity to be pear-shaped with the large end uppermost. The posterior wall, consisting of the psoas muscle and the fat pad, is inclined backward from below upward at an angle of about 50° and it forms a padded shelf which helps to support the organs of the upper abdomen. Pressure from above would be directed not downward in a straight direction but downward and forward toward the anterior abdominal wall at an angle of about 50°. It would strike the anterior abdominal wall at its lower portion whence it would be reflected backward toward the sacral hollow. (Coughing produces marked bulging of the anterior abdominal wall at its lower portion thus confirming the conception.) Because of the natural lumbar lordosis there is a recession of the pelvic cavity from that of the abdomen. This is rendered more conspicuous by the promontory of the sacrum. When there are marked and sudden rises of pressure within the abdomen as the result of strain or cough the visceral thrust from above is reflected by the lower abdominal wall into the pelvis and is directed downward and backward so that it impinges upon the superficial surface of the organs of the pelvic cavity far behind the aperture.

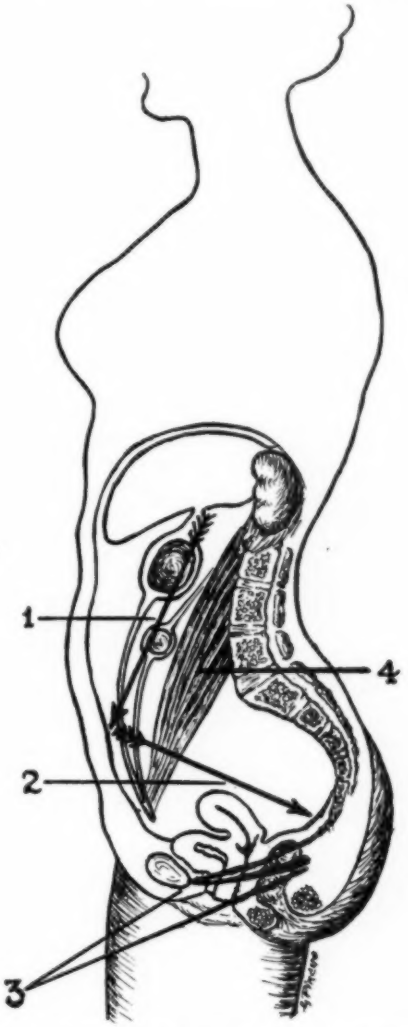

Fig. 3. Sagittal section demonstrating (1) direction of intra-abdominal pressure thrust; (2) deflection from abdominal wall into sacral hollow; (3) lower limit of pelvic floor aperture guarded by puborectalis muscle situated in most dependent portion of pelvis but anterior, out of direct line of deflected intra-abdominal pressure; (4) psoas muscle.

Thus, the pelvic floor aperture because of its anterior position (situated as it is immediately behind the symphysis pubis) is out of reach of the direct visceral thrust. In the erect posture, such a pressure wave can reach the aperture only by further reflection and diffusion. An added safeguard is the plasticity of the visceral mass. This further dissipates the force of the pressure wave causing it to diffuse. There-

fore despite its dependent situation in the pelvis, the aperture is really in an advantageous position. (Fig. 3.)

MAINTENANCE OF UTERUS IN ITS NORMAL POSITION

The uterus is maintained in its normal position by two factors. One is the interaction of the pressure of the intestinal coils from above exerted on its broad posterior surface against the supporting value of the pelvic floor musculature and the second is the volume of the connective tissue in which the pelvic viscera are embedded. An increase in the volume of the pelvic connective tissue such as occurs by the deposition of fat as part of a general obesity causes a relatively high position of the uterus in the pelvis.

Against the conception that the uterus is maintained in its position by the thickening of the connective tissue to which the cervix is attached and that relaxation of these connective tissue thickenings results in prolapse, are massed a group of facts. In the first place the normal mobility of the uterus and the restraining influence of the connective tissue are irreconcilable. If they fix the position of the uterus then the cervix should have less than its normal mobility. Since the cervix is mobile these tissues do not cause its fixation. If the mobility is due to slackness of these thickenings what causes the recession of the cervix to its normal position after release of the tension of a tenaculum by means of which it was drawn down to the vulva?

No matter how high the intra-abdominal pressure be raised voluntarily by straining, the cervix never descends to as low a level as it can be pulled by a tenaculum. When the uterus descends following straining, it does not do so alone. The downward movement is manifested in all its attachments, the other pelvic viscera, the embedding connective tissue and the pelvic floor itself. The reason why the uterus does not descend as far as it can be pulled by a tenaculum is that with the descent due to strain there is a synchronous, reflexly produced, increase in the tone of the levator preventing too much displacement and a more forceful closure of the pelvic floor aperture preventing extrusion.

These connective tissue thickenings are said to reach full development in adult life. Yet prolapse occurs in adult life and not in childhood. Infants are subjected to great rises of intra-abdominal pressures with frequent, sudden, down-thrusts of viscera, as in crying, but prolapse does not occur. Yet in infants with spina bifida in which the lesion involves the lower sacral nerves supplying the muscles of the pelvic floor, prolapse of the uterus is not an uncommon occurrence following the strain of crying even as early as the second or third day after birth.

DEVELOPMENT OF PROLAPSE

It becomes evident, then, that two interrelated causes are important in the production of prolapse. The first is an increased pressure in the pelvis, and the second, an insufficient resistance of the pelvic musculature. The exciting cause, the increased pressure in the pelvis, is one which is produced frequently, and often with marked variation in degree. Not infrequently it is the result of corpulency. This necessarily affects the pressure in the pelvis. When corpulency exists alone without the predisposing insufficient resistance of the pelvic floor musculature, the uterus rises high in the pelvis. When, however, it is found in conjunction with an incompetent resistance of the pelvic floor musculature, prolapse occurs.

The predisposing cause, the insufficient resistance of the pelvic floor musculature, is usually the result of childbirth injury. Such an injury affects most frequently the sphincteric mechanism through which the child passes, namely, the puborectalis muscle. The injury to the puborectalis muscle is most likely to occur in the anterior portion near the pubic attachment in the region of the bladder support

and not posteriorly where greater extensibility is possible. The result is a permanent enlargement of the aperture particularly in the transverse direction. As may be expected, the closure of the aperture in the anteroposterior diameter by approximation to the symphysis pubis is likewise interfered with.

Not every pregnancy is complicated by an injury to the puborectalis muscle. In many instances where it is injured the amount of injury is insignificant. In others, sufficient repair takes place during the lying-in period so that with the return to active life there is a resumption of functional activity sufficient to maintain adequate pelvic floor resistance to the intra-abdominal and intrapelvic pressure.

In the comparatively few instances where the pelvic floor resistance is insufficient as a result of atony of the muscle or general weakness, the whole musculature of the pelvic floor is involved. In those instances, the coccygei or the ileococcygei suffer as much as the puborectalis muscle. The greater the injury to the pelvic floor, the less will be the intrapelvic pressure rise necessary to produce prolapse. While hard physical manual labor would be insufficient to produce a prolapse in one with slight damage, even ordinary exercise may be responsible for extrusion of the uterus at the vulva in another with severe impairment and in the borderline cases where manual labor produces symptoms of prolapse it is easy to understand how a less arduous life often relieves them. Then, again, a woman in whom the injury to the pelvic floor, in relation to the amount of intrapelvic strain that she ordinarily undergoes is insufficient to produce prolapse, can develop a prolapse as time passes and the intra-abdominal pressure strains are increased by the gradual development of obesity.

When the pelvic floor aperture is enlarged as a result of injury to the sphincter mechanism, the bladder which rests above the aperture anteriorly and which is subjected to the forces of intrapelvic pressure is pushed down into the aperture everting the anterior vaginal wall in front of it. Thus, at first, it dilates the aperture and then it is extruded through it. In a similar manner dilatation of the anterior rectal wall because of increased pressure within, as a result of straining at stool occurring repeatedly in an individual in whom the aperture is larger, ultimately results in extrusion of the posterior vaginal wall overlying the rectum through the aperture with the consequent production of a rectocele.

Whether the bladder comes through the aperture first or the rectal wall begins to protrude first, matters but little. Ultimately, with the continuation of visceral thrust from above and an insufficient resistance on the part of the pelvic musculature, the cervix and uterus begin to slip through the pelvic floor aperture and finally present at the vulva. Thus, the anterior position of the cervix of which so much is made in the supposed antecedent retroversion is not the cause of prolapse. The uterus and cervix together are more or less non-deformable. As a result of intra-abdominal and intrapelvic pressure the anterior cul-de-sac becomes dilated with intestines and the uterus is pushed away from its anterior position where it rests on the bladder, back toward the sacral hollow and the cervix assumes a corresponding anterior position. It is to be expected that because the cervix is immediately over the aperture it frequently is the first organ to present itself at the vulva when the factors which produce prolapse have been active.

It is obvious that as the uterus becomes retroverted, the cervix anteverted and prolapse begins, elongation of the supposed supporting ligaments must occur. The attached connective tissue, the uterosacral and the pubovesical ligaments become greatly lengthened. The absence of elongation of these ligaments in normal individuals is due to the negligible tension to which they are subjected.

It is not an uncommon thing to find patients in whom the cervical connective tissue attachments are so loose that the uterus can be moved very freely in all directions on bimanual examination, yet who, leading active lives, have no signs or symptoms of prolapse because of a competent pelvic floor musculature. On the other hand it is inevitable that an active woman with an incompetent musculature and a considerably enlarged pelvic floor aperture will develop prolapse. Laceration or insufficiency of the pelvic floor aperture by itself cannot bring about prolapse. This in itself is but the predisposing cause. It is only when the patient begins to move about, undergoing varying degrees of strain, and performing long arduous manual labor that prolapse may arise, for then the exciting factors come into play.

The great mobility of the uterus in the normal individual is not due to the looseness of the connective tissue attachments but rather to the pliability of the visceral mass about the uterus. An increase in the volume of the visceral mass occasioned by distention of the bladder, the enlargement of the pregnant uterus, the growth of tumors in the pelvis leads to diminution in the mobility of the uterus. Another factor which becomes effective because of such diminished deformability of the viscera is the concomitant increase of the intra-abdominal and pelvic pressure. The increased intra-abdominal pressure factor tends to restrict the mobility of the viscera in their normal relationship.

In the normal individual, straining with marked increase of intra-abdominal and pelvic pressure results in a descent of the pelvic floor. There is no enlargement of the aperture however, because, reflexly, the tonicity of the muscles guarding the aperture is correspondingly increased so that with this exaggerated activity, no extrusion of pelvic viscera can occur. This synchronous muscular contraction can best be felt by the examining finger in a primipara at about the forth or fifth month because there the accommodation to the increasing size of the fetus and uterus with the concomitant increase in intra-abdominal pressure produces a hypertrophy of the pelvic floor musculature which lends itself more readily to the palpatory investigation of contractions. In an individual in whom there is a weakened, injured, dilated pelvic floor a similar strain would not only cause a descent of the floor but also an extrusion of the pelvic viscera with prolapse.

The normal respiratory rise and fall of pressure within the abdominal cavity is manifest on the peripheral surface of the body by a rhythmic motion downward and upward corresponding to the contraction and relaxation of the diaphragm. This can easily be observed on any patient when placed in the lithotomy position. When the diaphragm contracts and the intra-abdominal and intrapelvic pressure rises the pelvic floor (the levator ani muscle) is forced downward and with the relaxation of the diaphragm, it returns to its normal position again. During coughing, straining, sneezing, when there is a very marked rise in pressure in the abdomen produced not only by diaphragmatic contraction but also by marked contraction of the abdominal muscles, the normal non-lacerated pelvic diaphragm would be expected to bulge tremendously, and it would so bulge, were it merely an elastic structure without the possibility of contracting and without the regulatory reflex mechanism which insures its simultaneous contraction. It does not bulge to any greater extent because it does contract simultaneously with the abdominal wall and the thoracic diaphragm. This contraction can easily be palpated during coughing or sneezing and this contraction forms a very efficient mechanism for successfully withstanding the down-thrust occasioned by the sudden rise of intra-abdominal and intra-pelvic pressure. Were there not such a mechanism for withstanding the down-thrust, the pelvic viscera would certainly be extruded from the pelvis. It is not difficult to understand how such a mechan-

ism would be a much more effective restraint against visceral extrusion than would the radiating cervical connective tissue strands which can come into play only as their tension is increased by visceral descent and which even then are only capable of passive resistance. When such a patient is given spinal anesthesia vomiting movements may produce an extrusion of the cervix from the vagina because now the pelvic diaphragm no longer acts as a restraining mechanism because the anesthesia has temporarily interrupted the nervous mechanism which normally reflexly produces contractions of the levator simultaneously with intra-abdominal rises in pressure.

Where the pubococcygeus or puborectalis muscle is damaged, the visceral parts immediately adjacent to the pelvic floor aperture bulge into the aperture during the period of straining. The larger the aperture, the greater is the initial bulging, the greater the descent, and the more these parts can enter the aperture, the more is the expanding pressure exerted circumferentially on the boundaries of the aperture tending to enlarge it according to the amount of pressure transmitted to it from the pelvis. On the contrary, straining, while it affects those organs immediately over the pelvic floor aperture so as to attempt to extrude them, in the normal individual also causes equal pressure downward on the lateral boundaries of the aperture and because of the equality of the pressure transmission in all directions the tendency toward extrusion through the aperture is nullified by the pressure on the sides of the aperture which tends to close it.

In the case of a damaged pelvic floor musculature, when descent and bulging through the aperture occur, the aperture becomes secondarily dilated, allowing more and more visceral contents to come through. As the prolapse continues and the prolapsing organs descend they are grasped circumferentially by the contractions of the remainder of the pelvic floor. This continues until an equilibrium is established. This equilibrium depends especially upon the condition of the puborectalis. In the development of prolapse the puborectalis muscle hypertrophies as an expression of the attempt on the part of the individual to compensate for the original injury, that is, as an adaptive response. Occasionally, such a response may be sufficient to close the aperture and prevent further prolapse.

Besides prolapse due to obstetrical injury and to spina bifida in infancy, prolapse occurs in a very small group of nulliparous women (intravaginal hypertrophy of the cervix is not included in this group). These women are found to have spina bifida occulta with trophic disturbances in the pelvic floor musculature or constitutional pelvic floor musculature dystrophy. Thus the mechanism described fits the development of prolapse in all its phases.

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COMPLETE PROCIDENTIA IN THE AGED

OPERATIVE TREATMENT BY THE MODIFIED LEFORT TECHNIQUE

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THE pathetic figure of the aged woman suffering a complete utero-vaginal prolapse with all its attendant vesical and rectal discomforts is familiar to all. It is doubly pathetic when the patient has been told that she is too old for surgical and permanent relief. Perhaps the most important excuse for the writing of this paper is to refute the statement that any prolapse is so far advanced or any patient so decrepit as to contraindicate surgery.

The modified Lefort vaginoplasty is not generally nor widely known and yet when properly done is a veritable God-send to these sufferers. It is usually performed under local or spinal anesthesia. There is little or no shock. The operation has been successfully done upon a patient who was ninety-six years of age. It must be realized, however, that the operation suppresses the vagina, so that obviously it is reserved for those in whom sex relations are no longer to be considered.

Nothing original is claimed in this presentation. The writer wishes to confirm the excellent results previously reported by others and to describe graphically the technique. Ten additional cases treated and traced since 1930 are discussed.

The operation was first performed and described in 1877 by M. Leon Lefort of Paris.[1] Fanny Berlin of Boston[2] reported 3 cases in 1880 and I believe was the first American to do this operation. Casual perusal of American literature shows a marked paucity of case reports. Taft[3] reported a case in 1889; Erocq and Nora[4] 38 cases in 1925, and Phaneuf[5] 16 cases in 1931.

TABLE 1

Name	*Age*	*Duration of Prolapse*	*General Condition*	*Date of Operation*	*Anesthesia*	*Result*	*Follow-up*
M. S.	74	10 yrs.	B.P. 180/90. Nephritis	1930	Spinal	2–17–33	Good
C. M.	71	12 yrs.	B.P. 178/100 Nephritis. Cardiac	1930	Spinal	10–20–34	Good
N. R.	55	10 yrs.	B.P. 168/80. Diabetic	1931	Spinal	5– 3–34	Good
L. M.	84	5 yrs.	B.P. 190/100 Marked arterio-scl.	1932	Local	4– 1–34	Good
A. LeC.	62	5 yrs.	B.P. 210/110	1930	Spinal	4–1932. Deceased, apoplexy, no recurrence	
M. D.	63	?	Cardiac Involution psychosis	1930	Local	Transferred to Hudson River State Hosp. No recurrence 1932.	
M. J.	68	7 yrs.	B.P. 222/104 Diabetic	1932	Local	2–5–33 County Home reports good result.	
S. P.	74	?	B.P. 180/110 Slow fibrillator	1932	Local	10–5–33 County Home reports good result.	
C. R.	96	14 yrs.	B.P. 190/100	1933	Local	10–5–33 Result good.	
S. A.	68	9 yrs.	B.P. 105/65	1934	Avertin cyclopropane	6 mos. no recurrence	

PROCEDURE

Preoperative: The prolapse must be reducible. If reducible, bed rest and topical applications will so soften the presenting mass as to reduce it. It is imperative to clear up all decubitus ulcers prior to operation so as to insure primary union.

Anesthesia: Local or spinal anesthesia is preferable in this class of patients who are nearly always poor risks. The use of local infiltration facilitates dissection technique.

[1] LEFORT. New method for the cure of uterine prolapse. *Bull. Gen. de therap.*, 92: 337–344, 1877.

[2] BERLIN. *Am. J. Obst.*, 14: 866, 1881.

[3] TAFT, C. E. *Am. J. M. Sc.*, 98: 128 (Aug.) 1889.

[4] EROCQ, P., and NORA, G. *Gyn. & Obst. Per.*, 40: 339–355 (May) 1925.

[5] PHANEUF, L. E. *Mempbis M. J.*, 8: 145 (Oct.) 1931.

Details of Technique: (Figs. 1–5.) The principle involved is the denudation and subsequent coaptation of rectangular areas on the anterior and posterior vaginal walls respectively, thus creating a wide median supporting bar, above which rests the cervix. This bar is about 4 cm. by 8 cm. in the average case and forms very effective support for the cervix and uterus. To either side of this median bar is a small canal completely invested by vaginal mucosa and which carries off any cervical secretion. A high perineorrhaphy completes the operation and is a necessary part of it. A preliminary currettage is done to preclude fundal malignancy. Erosion or ulcer of the cervix is taken care of by cautery or amputation.

Fig. 1.

Fig. 2.

Fig. 1. Cervix delivered entirely outside of body, placing anterior vaginal wall on stretch. Proposed rectangular denudation outlined by scalpel and denudation about 4 cm. wide and 8 cm. long.
Fig. 2. Corresponding rectangular denudation completed on posterior wall.

REPORT OF CASES

The cases operated on at Grasslands and Mount Vernon Hospitals (Table 1) are presented as examples of what can be easily accomplished by this operation. The youngest patient was fifty-five, the oldest ninety-six and the average sixty-eight years of age. All patients had some constitutional disease or infirmity. No mortality or recurrences are noted to date.

SUMMARY

An attempt has been made to recall the attention of the profession to the operation of Lefort with slight modifications in technique. It is hoped that its use will become more widespread for the relief of these aged and many times decrepit individuals. Ten additional cases are reported. It is the opinion of the writer that for complete procidentia in the aged no operation is as successful and at the same time so free from danger as that of Lefort.

My thanks are due the artist, Miss Florence Nathan.

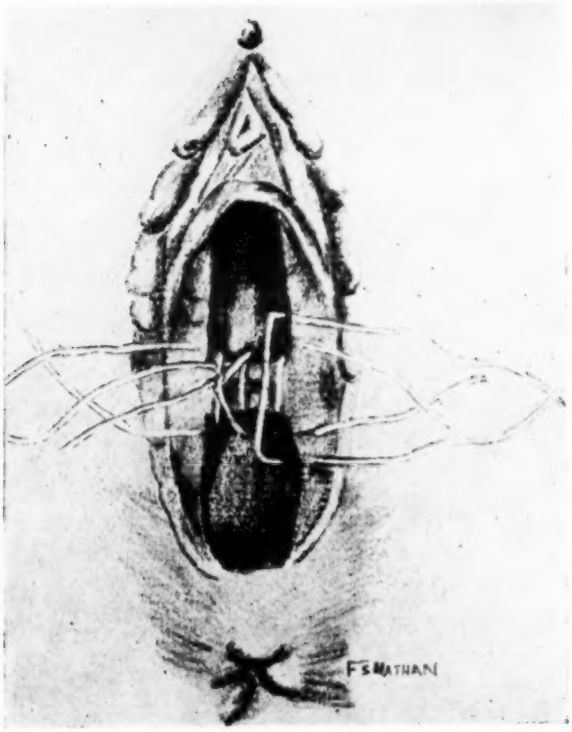

FIG. 3A. Anterior and posterior denudation completed. Placing of sutures begun.

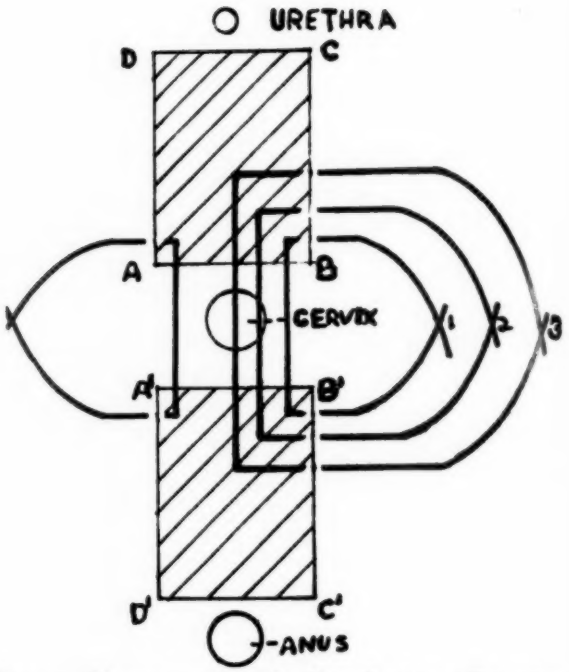

FIG. 3B. Diagrammatic and self-explanatory. By means of sutures correctly placed two denuded areas are approximated, A TO A, B TO B, C TO C, etc.; thus gradually pushing cervix upward and eventually approximating anus to meatus. Six or seven sutures on either side usually suffice. An occasional midline suture to obliterate dead space is advisable. Sutures must be accurately placed so as to keep lateral canals completely invested by mucous membrane.

FIG. 4.

FIG. 5.

FIG. 4. Prolapse replaced and all sutures tied. Lateral canals are shown as well as medium bar with some exaggeration in size for better illustration. A high perineorrhaphy of the Hagar type is being made.

FIG. 5. Completed operation. Cervix high up, a median bar about 1" wide separating two narrow vaginal canals, one to either side, completely invested by mucous membrane and extending to cervix to carry off any secretions. High perineorrhaphy to buttress median septum.

A STUDY OF

DIAGNOSTIC ERRORS IN FOUR YEARS OF ADMISSIONS TO A GYNECOLOGICAL SERVICE*

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IN September of 1929, a blank volume was set aside on the gynecological service of the Harlem Hospital and was named, the "Calamity Book." In it, from the date of its inception, were placed extensive abstracts of those cases in which death, complication, wound infection or diagnostic error occurred. Cases of unusual interest, although not calamitous, were also abstracted and included. In the four year period from September 1, 1929 to September 1, 1933, 2810 cases were admitted to the Service and ultimately classified as diagnosed or undiagnosed. When an opinion was proved at operation, by biopsy, by prolonged clinical study, or by necropsy, the diagnosis was considered proved. Where no diagnosis could be established, the case was classified as undiagnosed. Cases admitted to the Service and later transferred to other services were not considered in the diagnostic classification.

In the series of 2810 admissions, proved diagnoses were as follows:

	Cases	*Per Cent*
Abortions (threatened, complete, incomplete, etc.)	840	29
Fibroids	418	14.5
Chronic Salpingitis	370	13
Pyosalpinx or tubo-ovarian abscess	351	12.5
Acute salpingitis	119	4.4
Ectopic Gestation	87	3
Ovarian cysts (including dermoids)	69	2.7
Pelvic abscess	59	2
Pregnancy	46	1
Carcinoma of the cervix	40	1

In the series there were 61 other gynecological entities but these were all less than 1 per cent of the total series.

In the groups of cases, entirely unselected and listed as they appeared in order of admission, 43 per cent fell into the abortion and fibroid group. Perineal lacerations and pelvic floor relaxations comprised less than 1 per cent of the total series. It has been characteristically the experience of the service to find birth injuries playing a very minor rôle in the number of admissions. The fact that the greater percentage of admissions to the service is colored may explain the great incidence of fibromyomata.

In 2810 admissions, diagnostic error occurred 68 times, giving a percentage of error of 2.3. Startlingly low though this may seem, it is readily apparent that there are numerous reasons for its minuteness. Primarily, gynecological diagnosis is extremely limited; secondly, abortions present themselves for admission with diagnosis established; thirdly, the type of fibroid seen on the Service is easily distinguishable. Reference to the figures will indicate that approximately 43 per cent of cases present no diagnostic problem whatsoever. Even in those cases which produced the highest percentage of diagnostic errors (ectopic gestation), the picture presented on admission by the average case was the readily recognizable "tragic state" with shock, extreme pallor, thirst, air hunger and the typical history.

Considering these factors, (and there are others) the small figure of 2.3 per cent looms menacingly large.

Sixty-eight cases of diagnostic error are classified as follows:

Ectopic gestation	27
Fibroid uterus	24
Intraligamentous disease	4

* From the Gynecological Division of the Surgical Service of the Harlem Hospital, N. Y. C.

Ovarian cyst	3
Tubo-ovarian abscess	3
Acute appendicitis	3
Ovarian cyst with twisted pedicle	2
Pelvic tuberculosis	2

Before venturing to a brief discussion of the erroneous diagnoses, the method employed in registering errors concerned with ectopic gestation must be noted. The service records ectopic gestation, in so far as diagnosis is concerned, as follows:

1. Diagnosis made and found
2. Diagnosis made and not found
3. Diagnosis not made and found.

Ectopic gestation is too often diagnosed without being demonstrated at operation. Probably the major portion of the errors in the diagnosis of ectopic gestation are of this character. Reports rarely suggest this to be the case. Further, it must be noted that there is no difficulty in the recognition of the "bled-out" ectopic. Such cases, as a rule, present the textbook picture. The greatest pitfalls lie in the recognition of those cases wherein the true pathology is that of tubal or missed tubal abortion.

Ectopic gestation was concerned in 27 diagnostic errors in this series. In 13 cases the diagnosis *was not* made and ectopic gestation was found at operation. The preoperative diagnoses were:

1. Infected intraligamentous cyst
2. Pyosalpinx
3. Tubo-ovarian abscess
4. Ovarian cyst with twisted pedicle
5. Fibroid uterus and dermoid cyst
6. Infected abortion
7. Fibroid uterus
8. Pyosalpinx
9. Bilateral pyosalpinx
10. Appendiceal abscess
11. Pelvic abscess
12. Acute appendicitis
13. Pyosalpinx and salpingitis.

In 14 cases, a preoperative diagnosis of ectopic gestation was made and ectopic *was not* found. Operation revealed the following pathology:

1. Bilateral pyosalpinx
2. Ruptured uterus; ruptured gut
3. Ovarian cyst
4. Parametritis
5. Omental tumor
6. Gangrenous colitis
7. Bilateral ovarian cysts
8. Tubo-ovarian abscess
9. Ruptured pyosalpinx
10. Bilateral pyosalpinx
11. Acute salpingitis
12. Ruptured lutein cyst
13. Bilateral pyosalpinx
14. Intestinal obstruction.

Fibroid uteri were concerned in 24 cases of diagnostic error. In 18 cases the diagnosis of fibroid uterus was made preoperative and the following pathology was revealed at operation:

1. Bilateral ovarian cyst
2. Tubo-ovarian abscess and pyosalpinc
3. Pyosalpinx rupturing through the anterior abdominal wall
4. Pregnancy and ovarian cyst
5. Terminal ileitis
6. Bilateral dermoid cysts
7. Intraligamentous cysts
8. Placenta accreta
9. Multiple fibroids and pregnancy
10. Hydrosalpinx
11. Ectopic gestation
12. Pregnancy
13. Bilateral pyosalpinx
14. Abscessed lutein cyst
15. Tubo-ovarian abscess
16. Solid tumor of the ovary
17. Bilateral pyosalpinx
18. Ovarian cyst.

In 6 cases fibroid uteri were found at operation with the preoperative diagnoses:

1. Bilateral pyosalpinx
2. Ovarian cyst
3. Dermoid cyst
4. Tubo-ovarian abscess
5. Bilateral pyosalpinx
6. Ovarian cyst.

Intraligamentous pathology was concerned in 4 cases of error. In those cases such pathology was demonstrable at operation but the preoperative diagnoses were:

1. Bilateral pyosalpinx and ovarian cyst
2. Ruptured ectopic gestation
3. Fibroid uterus
4. Bilateral pyosalpinx.

Ovarian cyst was concerned in diagnostic error in 3 cases. Preoperative diagnoses in these cases were pyosalpinx (2) and mesenteric cyst (1) and at operation ovarian cysts were demonstrated.

In 3 cases a preoperative diagnosis of tubo-ovarian abscess was made and operation revealed:

1. Bilateral pyosalpinx
2. Papillary cyst-adenocarcinoma of ovary
3. Pyosalpinx and ovarian cyst.

In 3 cases diagnosed as acute appendicitis, acute salpingitis was found at operation.

In 2 cases, ovarian cyst with twisted pedicle was diagnosed and operation proved one to be a ruptured ectopic gestation and the other a bilateral parametritis.

Pelvic tuberculosis was twice erroneously diagnosed. In one case, hydrosalpinx with prevesical and postvesical abscesses was demonstrated and in the second case, pelvic tuberculosis was present but the case was considered to be a pelvic abscess.

It is impossible to refrain from remarking on the rarity of pelvic tuberculosis on the service, this in light of the fact that the overwhelming majority of the cases are of the colored population. The clinical diagnosis of the condition is difficult.

The comparison between this series of diagnostic errors and the small total percentage of errors throws new light on that low figure. The comparison between preoperative and postoperative diagnoses is startling and one well wonders how such errors were committed. The inevitable fact remains; the false opinions were arrived at and placed enduringly in the hospital records. These opinions were not those of the House Staff but of visiting men apparently well trained in diagnosis. In 54 of the 68 cases the entire staff expressed incorrect opinions. In 14 cases some one staff member made the correct diagnosis.

With 54 cases of diagnostic error registered in the "Calamity Book" an analysis of these cases was undertaken with an eye to discovering the possible cause of error. The charts were examined in detail in an attempt to determine those factors which led to the formation of erroneous opinion. In 39 cases, despite the ease with which retrospective diagnosis is made; despite the towering advantage of calm reflection; despite the presentation of an entire clinical course and study, no logical medical reasoning leading to the correct diagnosis could be prepared. In 10 of these cases, by devious windings and tiresome pursuit, the truth is reached but the imagination becomes wearied. In 2 cases, carelessness on the part of the House Staff was the cause of the downfall of opinion.

In only 17 of 54 cases, the past yielded the truth; charts readily demonstrated irrefutable evidence which fairly shouted the correct diagnosis. Out of them, the Service has gleaned what might be termed axioms in gynecological diagnosis. The ultimate divining rod in diagnosis is the pelvic examination. In this series of diagnostic errors, it has been found that the description of pelvic pathology by means of the vaginal touch picture is a tremendous variant. It is a rarity to find two examiners arriving at a similar description. Variations occur not only in the estimation of the size and consistency of the pelvic contents but even in location. It is not uncommon to find one examiner describing a mass where another found none. It is no rarity to find one report stating a mass to be solid and the next to term it cystic.

Vaginal examination under narcosis remedies this situation to a great extent. The narcotized patient is fully cooperative. There is offered no resistance to the examiner and he may prolong his examination as he desires, carefully and completely exploring the pelvis without causing distress or pain. Repeated examination under narcosis is well nigh a guarantee of better anatomical diagnosis.

The value of accurate gynecological diagnosis may well be disputed. There are those who rarely seek to establish anatomical description of pathology. The presence of a pelvic mass may be sufficient indication for therapy, surgical or of other type. This is slipshod. It is unfair to the patient. At times it is no light matter. It may become embarassing in the extreme to open an abdomen and find a pregnant uterus which was not predicted. It may be lethal to burst into an undiagnosed and unsuspected parametritis. Beyond scientific demand for accuracy there is the pride in proved opinion and there is the diagnostic record of a service to be considered. Such volumes as the "Calamity Book" are available to any interested reader.

From the errors of the past, certain facts have been elicited. These, if considered in future cases presenting diagnostic problems, may prevent error. Each has been derived from one or more cases in which it pointed the way to correct diagnosis, but was, in some manner, overlooked. Out of the "Calamity Book" the following conclusions are drawn and presented:

1. Where there is elicited a history of irregular menstruation, every detail of that irregularity must be determined, to disregard menstrual irregularity is to invite error in the diagnosis of ectopic gestation.

2. In eliciting the menstrual history, mere regularity is by no means sufficient. The past menstrual history must be known and the absolute norm ascertained, before anomalies may be considered to exist.

3. Where ectopic gestation is suspected and vaginal bleeding or discharge is present, its exact nature must be determined either by close questioning or by direct examination of the discharge. The vaginal discharge in ectopic gestation is almost pathognomonic.

4. In the history of ectopic gestation, a previous period of sterility is of minor importance. Of far greater importance is a definite history of pelvic infection. Where a history of currettage is obtainable it may prove to be of great importance.

5. In the blanched, bled-out case, where the blood pressure is normal or elevated, acute hemorrhagic states need not be considered.

6. Intraligamentous pathology is rarely, if ever, correctly diagnosed. This should be borne in mind when the diagnosis is suggested.

7. Inflammatory cysts are never diagnosed on vaginal examination yet at operation, one sees readily how they constantly confuse the vaginal touch picture.

8. Where all factors point in one direction and the sedimentation rate takes a diametrically opposite diagnostic path, follow the sedimentation rate. To disregard it is to invite error.

9. After the stethoscope has ruled out the fetal heart and the catheter has emptied the bladder, the huge abdominal mass may be interpreted as a giant fibroid or ovarian cyst or tumor. The differential diagnosis between the two is by no means a sinecure.

10. In the face of an amenorrhea and an enlargement of the uterus no diagnosis is tenable until the presence of pregnancy is proved or disproved.

11. Where pregnancy is suspected, no surgery is indicated until laboratory tests (*x*-ray or Aschheim-Zondek test) prove or disprove its presence.

12. Pelvic diagnosis following previous pelvic surgery is dangerous. Most dangerous of all is the diagnosis of fibroid uterus following previous laparotomy.

13. The words "possible," "probable," and the phrase "to be excluded," confuse the diagnostic issue.

14. Change of diagnosis because of findings in a preoperative examination under narcosis is dangerous. It precludes consideration of the history and clinical course.

15. Change of opinion must be made only on the virtues of fresh evidence obtained through the laboratories or by prolonged clinical observation.

16. The clinical sense will never be demonstrated but it is a very actual thing.

The apparently snap, unfounded judgment of the experienced observer is often the solution to a perplexing diagnostic problem.

17. In the emergency case, the visiting surgeon should be presented with the patient and all available laboratory data. He should elicit the history himself.

18. The house surgeon should be directly responsible for all laboratory findings in questionable cases.

The Service permits itself no smug complacence because of its policy to recognize and report diagnostic errors. Dust collects over the best intentions. To avoid the possibility of disregard of the lessons to be learned from past errors, it is the plan of the service to analyze all errors each six months. In such a manner the accuracy in diagnosis may be improved. In such a manner the occasional accident associated with diagnostic error may be prevented.

SUMMARY

It has been the purpose here to present those facts which have been obtained through prolonged study of error in gynecological diagnosis as recorded in complete case histories. It is felt that such facts, almost axiomatic in nature, may serve as efficient prophylaxis against repetition.

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[Continued on p. 322.]

* Continued from p. 194.

EXTRA-UTERINE PREGNANCY FOLLOWING THE INJECTION OF IODIZED OIL FOR UTEROSALPINGOGRAPHY

ETIOLOGY AND PREVENTION SUGGESTED: WITH REPORT OF A CASE*

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THE injection of iodized oil for uterosalpingography is an established procedure of great value; but it is not entirely a harmless one. Novak[1] cites a case in which the contrast material was still present after fifteen months and advises the abandonment of this method of diagnosis and treatment. Morhardt[2] states that some authors think that the use of iodized oil for uterosalpingography can disturb the function of the tubes by producing an irritation or inflammation of the tubal mucosa which either prevents the ascent of the spermatazoa or the descent of the ova, in the latter case favoring ectopic pregnancies. While 4 cases of extrauterine pregnancies after uterotubal insufflation have been reported (Rubin[3] reports 3 cases; Elinek[4] reports one case), I have been unable to find any report of an extrauterine pregnancy following the injection of iodized oil for uterosalpingography. The following report should for that reason be of general interest.

REPORT OF A CASE

CASE 1. Mrs. S. K., aged twenty-six, married three years, nulliparous, presented herself for examination in June, 1929. Her chief complaint was sterility. Her husband had been married before. He was the father of two children by his first wife. Examination of his seminal fluid revealed the presence of many active, normal spermatozoa. A uterosalpingography was done, iodized oil being used as the radiopaque medium. No manometer was used and the procedure was accompanied by cramp-like pains just above the pubis. Figure 1 discloses the antero-posterior view after the injection of 5 c.c. of the iodized oil. It indicates that bilateral obstruction existed at both fimbriated ends of the tubes. The twenty-four-hour plate (Fig. 2) indicates that a little oil got through into the pelvic cavity on both sides, more on the left side than on the right side; a pool of oil may be seen imprisoned at each fimbriated end, more being imprisoned on the right fimbriated end than on the left. In May 1931, twenty-three months after the uterosalpingography, the patient was operated upon for a right-sided extrauterine pregnancy.

DISCUSSION

A. INCIDENCE OF EXTRA-UTERINE PREGNANCY

(1) *In the Pre-uterotubal Insufflation and Uterosalpingography Era: Shumann,*[5] in 1924, after a very careful analysis, determined that there was one extrauterine pregnancy for every 300 normal pregnancies. Urdan,[6] in 1930, analyzed 474 cases of extrauterine pregnancies which occurred at Mt. Sinai Hospital, New York City, over a nineteen-year period. He placed the incidence at about 1.5 per cent. These figures represent the extremes found in the literature. Urdan found that primary (10 per cent) and secondary (12 per cent) sterility accounted for 22 per cent of the cases of extrauterine pregnancy. We may thus arrive at the ratio of one extrauterine pregnancy due to sterility to every 500 to 1500 cases of normal pregnancies.

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(2) *In the Post-utero-tubal Insufflation and Uterosalpingography Era:* The only statistics available are those of Rubin.[3] He reports an incidence of 3 extrauterine pregnancies after uterotubal insufflation in 2000 cases of infertility. In this series there were 205 intrauterine pregnancies. This makes a ratio of one extrauterine pregnancy in 667 cases of sterility. It also makes a ratio of one extrauterine pregnancy in 68 intrauterine pregnancies resulting from uterotubal insufflation in this group of infertility cases. From these figures, which must of necessity be evaluated in accordance with one's philosophic turn of mind, it appears that uterotubal insufflation carries with it a somewhat greater chance of resulting in an extrauterine pregnancy than if no interference was practiced. On the whole, the results of uterotubal insufflation are so satisfactory that one should only bear in mind (1) that extrauterine pregnancy may occur, (2) that etiologic possibilities should be searched for and understood, and (3) that certain preventive measures may thereby be taken.

Fig. 1. Case 1. Antero-posterior view after injection of 5 c.c. of iodized oil. It demonstrates bilateral obstruction at fimbriated ends of tubes.

No comparable statistics are available, so far as I know, for determining the incidence of extrauterine pregnancy after the injection of iodized oil for uterosalpingography. Robins and Shapira,[11] in 1931, reported 32 pregnancies after 133 cases of sterility studied by uterosalpingography. Greenhill,[12] in 1933, culled from the foreign literature 70 cases of sterility, with 15 pregnancies resulting. In my own series of 12 cases of sterility studied by uterosalpingography over a period of six years (analyzed further below) there were 3 normal pregnancies and one extrauterine pregnancy. Thus out of 215 cases so studied there were 50 normal pregnancies (23.2 per cent) and one extrauterine pregnancy. It would appear from these figures that there is slightly more danger of abnormal gestation following the use of iodized oil for uterosalpingography than when uterotubal insufflation is done. However, this danger appears to be more than offset by the increased number of normal pregnancies (at least twice as many) following the use of iodized oil for uterosalpingography.

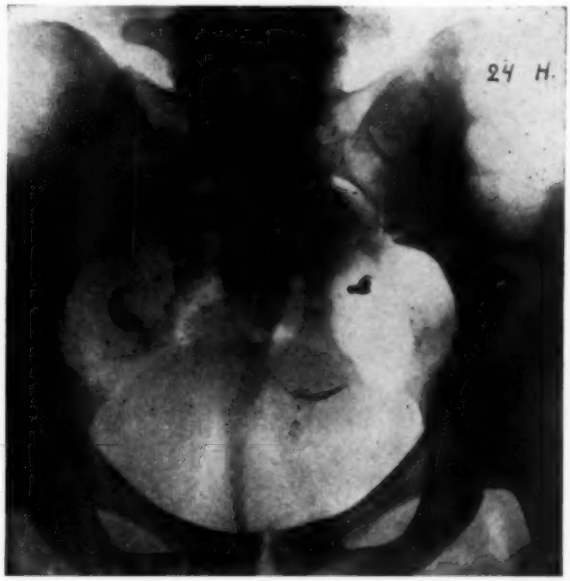

Fig. 2. Case 1. Twenty-four-hour plate which demonstrates that a little oil got through into pelvic cavity on both sides, more on left side than on right. It also demonstrates pools of oil imprisoned at each fimbriated end, more being imprisoned on right than on left.

B. ETIOLOGY OF EXTRA-UTERINE PREGNANCY FOLLOWING THE INJECTION OF IODIZED OIL FOR UTEROSALPINGOGRAPHY

It is conceivable that injection of iodized oil for uterosalpingography shortly after coitus may result in extrauterine pregnancy. The same reasoning quoted by Elinek[4] and Rubin[7] applies here. Spermatozoa may be washed into the peritoneal cavity with subsequent extra-uterine impregnation of the ova taking place. Then, too, irritation of the tubal mucosa may offer a barrier to the impregnated ovum (Morhardt[2]), as may endometrial implants (Elinek,[4] Rubin[7]). Furthermore, the long retention of the oil may also be a factor (Novak[1]). Figures 3 to 6 illustrate that encapsulated or inspissated oil may be present in the peritoneal cavity for as long as twenty-one months after uterosalpingography.

C. PREVENTION OF EXTRAUTERINE PREGNANCY FOLLOWING THE INJECTION OF IODIZED OIL FOR UTEROSALPINGOGRAPHY

(1) This procedure should not be performed in the presence of (a) active infection, (b) hemorrhage, or (c) normal pregnancy.

(2) The most favorable time is from four to seven days after the menstrual period, as recommended by Rubin[7] for the procedure of uterotubal insufflation.

(3) Pressures greater than 200 mm. Hg should not be employed. If no manometer is used, the injection should be stopped when the patient complains of cramp-like pains.

(4) Only small amounts of oil should be used. I feel that no more than 7 c.c. need be injected for all purposes.

(5) *Transuterine Injection of Amniotic Fluid to Wash Out the Oil and to Prevent Reformation of Adhesions (A Procedure Which May Also Be Used after Uterotubal Insufflation):* Because of my experience with the extrauterine pregnancy just reported I am now washing out the tubes with amniotic fluid.[8] I suggest the following technique:

(a) *When a Radiopaque Oil Has Been Used and One or Both Tubes Have Been Demonstrated to Be Patent:* Not more than 10 c.c. of amniotic fluid is introduced into the uterus under gentle syringe pressure with a Keyes urethral nozzle (as with the Jarcho[9] technique for the administration of iodized oil) immediately after the twenty-four-hour plate has been taken.

(b) *When Uterotubal Insufflation Has Demonstrated that One or Both Tubes Is Patent:* Immediately after the insufflation, while the patient is still on the table, 10 c.c. of amniotic fluid are injected into the uterus as above.

(c) *Case Illustrating the Slow Absorption and Encapsulation of Iodized Oil and in Which Amniotic Fluid Was Used to Maintain Tubal Patency:*

Case II. May 19, 1931: A uterosalpingography was done and left tubal obstruction diagnosed (Fig. 3).

May 20, 1931: A twenty-four-hour plate was taken. This exhibited the oil distributed mainly on the right side of the pelvis (Fig. 4).

June 19, 1931: A plain plate was taken which demonstrated a goodly portion of the oil still imprisoned among adhesions across the pelvis. There could be no assurance that some of this oil was not blocking one or both tubes (Fig. 5).

August 14, 1932: Having learned of the extrauterine pregnancy already reported and hoping to prevent another in this case, I injected 30 c.c. of amniotic fluid into the pelvic cavity via the transuterine route.

November 27, 1931: Tubal patency was obtained by insufflation of carbon dioxide after three trials at 200 mm. Hg. At the fourth trial the pressure dropped to 150–140–120 and finally to 95 mm. Hg. Partial obstruction of the tubes was diagnosed by Dr. G. G. Ward, of New York City, whom we had consulted. He advised another insufflation after the next period if pregnancy did not occur.

February 25, 1932: Again, on my own initiative, instead of insufflation, I introduced 15 c.c. of amniotic fluid into the pelvic cavity via the transuterine route.

February 10, 1933: A plain plate shows the presence of encapsulated or inspissated oil

in the right vertebral gutter (Fig. 6). This was pointed out to me by Dr. I. C. Rubin, of New York City, whom we consulted the following day.

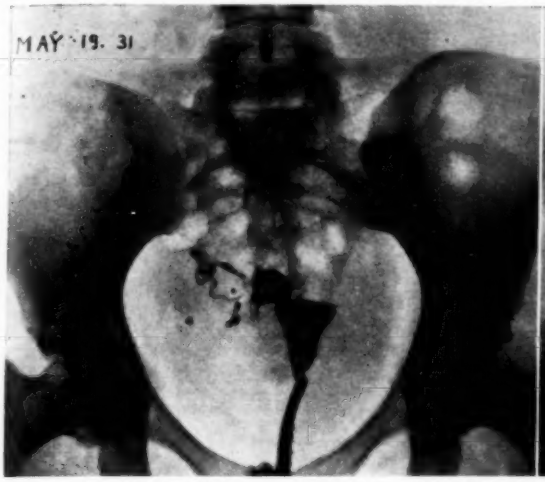

Fig. 3. Case 11. This demonstrates left tubal obstruction. Note tubal sphincter at left cornu. Without twenty-four hour plate (Fig. 4) this might be considered a physiologic obstruction.

Fig. 4. Case 11. Twenty-four-hour plate. This exhibits oil mainly distributed on right side of pelvic cavity. I am indebted to Dr. J. Jarcho, whom we consulted shortly after this plate was taken, for the interpretation. Distribution of oil mainly on one side indicates at least partial obstruction of opposite tube. Blobs of oil in center are representative of oil imprisoned among some adhesions.

February 11, 1933: Tubal insufflation was done by Dr. I. C. Rubin who reported to me as follows: "The kymographic tracing of the uterotubal insufflation determined patency within normal limits but also pointed to the presence of peritubal adhesions."

April 23, 1934: I delivered the patient of a normal male infant, the pregnancy having occurred twenty-six months after the introduction of iodized oil for uterosalpingography.

D. Pregnancy Follows Long After the Injection of Iodized Oil for Uterosalpingography

Over a period of six years I have performed 13 uterosalpingographies in 12 cases. I have been able to trace 11 of the cases. The twelfth case is counted in because the uterosalpingography disclosed bilateral obstruction at the cornua. While I appreciate that such obstruction may be obtained by physiologic constriction of the "cornual spincters" (Jarcho[10]), and the woman may therefore subsequently have become pregnant, I am counting the case as one in which pregnancy did not occur.

There were 4 pregnancies following the uterosalpingographies in the 12 cases. The first one, a case of secondary sterility, became pregnant immediately after the uterosalpingography. The second one, here reported, developed an extrauterine pregnancy twenty-three months after the uterosalpingography. The third one, also reported here, had a normal pregnancy twenty-six months after the uterosalpingography. The fourth became pregnant twenty months after the uterosalpingography. She is now in the seventh month of what appears to be a normal gestation.

This delay in impregnation following the use of iodized oil for uterosalpingography is in striking contrast to the rapidity of impregnation when that occurs following uterotubal insufflation. However, the incidence of pregnancy following the use of iodized oil appears to be two or three times that following uterotubal insufflation.

E. General Comment

From a consideration of my own cases and from those culled from the literature I have come to the conclusion that insuffla-

tion with gaseous products may successfully result in tubal patency. However, in cases of partial obstruction, such a procedure does not prevent the collapse of the tubes with the consequent reformation of intratubal adhesions as soon as such gases are absorbed (a matter of less than a day when judged by the cessation of shoulder pain). Radiopaque oils may be, by their slower absorption, more successful than gases in the maintenance of tubal patency. Here again, however, it is conceivable that, by its slower absorption, inspissation or encapsulation (Jarcho[10] and my own case Figs. 3–6) of the oil within or about the tubes may occur and thus defeat the purpose for which it was introduced.

To overcome these possibilities it occurred to me that amniotic fluid (which I had been instilling intraperitoneally for some years in my operative work, and which had given me the impression that my postoperative morbidity was considerably lessened thereby), by its ability to stimulate repair and prevent the reformation of adhesions, would be the ideal medium for maintaining tubal patency once established by other means.

I am compelled, too, by the experience with the extrauterine pregnancy here reported, to conclude that the uterotubal injection of iodized oil is not entirely harmless. It appears that the oil is not only slowly absorbed and may, by remaining in the tubes for as long as two years retard the advent of pregnancy, but such delay in absorption may be also a factor in the development of extrauterine pregnancies. In the case of extrauterine pregnancy here reported, it is conceivable that the oil became encapsulated at the right fimbriated end (Fig. 2). Slow absorption of the oil occurred, with partial patency of the tube resulting, but not sufficient patency to permit the impregnated ovum to reach its normal place of nidation within the uterus.

Fig. 5. Case 11. One month after uterosalpingography. This demonstrates that a goodly portion of oil is still imprisoned among adhesions across pelvic cavity. There can be no assurance that some of this oil was not blocking one or both tubes.

Fig. 6. Case 11. Twenty-one months after uterosalpingography. This plate was interpreted by Dr. I. C. Rubin, whom we consulted the following day. He pointed out presence of encapsulated oil in right vertebral gutter. Arrows delimit upper and lower poles of oil.

SUMMARY

A case of extra-uterine pregnancy following the injection of iodized oil for uterosalpingography is reported. The incidence of extrauterine pregnancies before and after the advent of tubal patency procedures is reviewed. Etiologic factors are discussed and preventive measures are suggested, especial attention being drawn to the use of amniotic fluid for maintaining tubal patency.

[For Bibliography see p. 310.]

EXPERIMENTAL TRANSPLANTATION OF THE OVARY

AUTOTRANSPLANTS AND HOMOTRANSPLANTS OF DOG OVARY INTO OMENTUM (PRELIMINARY REPORT)*

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SINCE Knauer's report, in 1896, of the first successful autotransplants of ovaries in animals, much work has been done on transplantation of the ovary by investigators in this country and in Europe. There naturally followed attempts to obtain viable and functional homotransplants and heterotransplants. In 1901, Foá and others reported that homotransplantation of the ovaries could be carried out successfully, but that autotransplantation is far more likely to be successful. Heterotransplants as a rule are failures. Thorek, in 1930, reported a successful heterotransplant of a human ovary into a monkey's neck. The transplanted tissue was viable at the end of eleven months.

Clinically, successful cases of autotransplantation and homotransplantation of the ovary have been reported by Morris, Marshall and Jolly, Martin, Thorek, Norris and Behney, Foster, Solomons, and others. Various sites have been used for the ovarian implants: the kidneys (Marshall and Jolly), the retrorenal space (Thorek), the omentum (Cotte and Colson), the uterus (Petit, Serdyukov, Kakuschkin), the rectus muscle (Norris and Behney), and the labia majus (Nard, Rouhier).

Experimentally, attempts have also been made to stimulate the growth of ovarian transplants by the administration of various endocrine products: pituitary extract (Engle, Haterius and Nelson, Goecke and Wirz), folliculin (Laprida), and thyroid extract (Madruzza). Cotte and Colson used insulin for this purpose in one of their cases.

It is clear that up to the present time there has been no uniformity as to results.

Recently, we have been carrying out a series of experiments upon dogs with the purpose of further investigating the possibilities of the omentum as a site for ovarian autotransplants and homotransplants. The omentum would seem to be an ideal site because of its proximity to the ovary, its striking powers for rapidly increasing its blood supply, its capacity for rapid absorption, and its remarkable reaction to infectious processes. The striking increase in blood supply is well illuminated in the cases of parasitic myoma, described by Kelly and Cullen.

In this preliminary paper we wish to report: (1) the results of autotransplants as observed at the end of forty-five and one hundred five days; (2) homotransplants as studied after forty-five days, and (3) one observation upon a pedicle autotransplant made ninety-one days after implantation. In a later report, the results of studies made six and nine months after implantation will be given.

EXPERIMENT I

On January 17, 1934, the right ovary of a dog was exposed under aseptic conditions. The bursa ovarii was opened and the ovary was freed from its attachments, bisected, and was fixed to the right side of the omentum with fine black silk, in such a fashion that it was completely enveloped by omentum. On May 1, 1934, the ovary was removed. The histologic section (hematoxylin-eosin stain) shows viable and functioning ovarian tissue

* From the Surgical Hunterian Laboratory, Johns Hopkins University.

(Fig. 1). Graafian follicles are found in all parts of the stroma except near the hilum. The photomicrograph of this section

EXPERIMENT 2

On January 17, 1934, the left ovary of a dog was exposed as in experiment 1. The

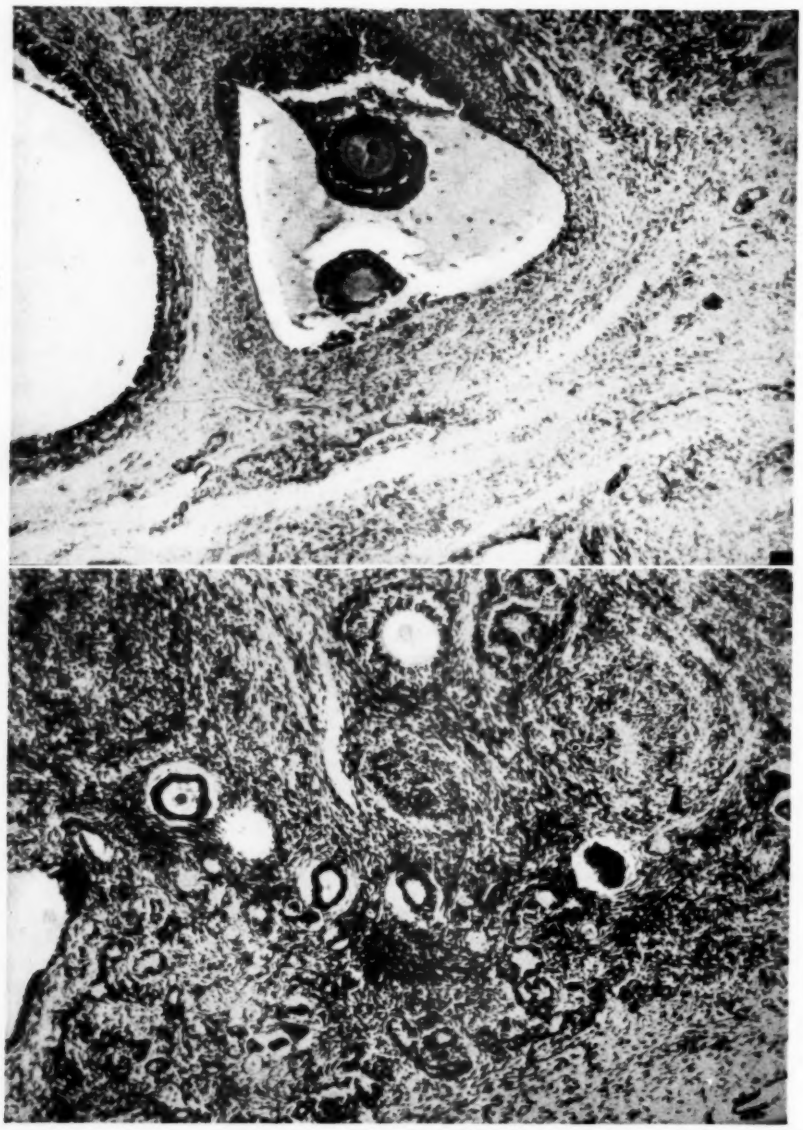

Fig. 1. Photomicrograph showing two ova in a single graafian follicle. On left is another follicle which is larger and is somewhat cystic. Ovarian autotransplant one hundred and five days old.

Fig. 2. Photomicrograph showing many follicles in all stages of development. Note numerous atretic follicles. Ovarian pedicle autotransplant ninety-one days old.

shows one graafian follicle containing two ova, and the detail of the discus proligerus and the theca externa and interna, although the latter does not show up very clearly in the reproduction. In one corner of the section, the stroma appears more condensed. This is because it is very near the tunica albuginea. There are a few scattered lymphocytes. The age of this implant is one hundred five days.

left border of the omentum was brought down and sutured to the left ovary and its mesosalpinx with fine black silk. On January 31, 1934, the left ovarian vessels were clamped, divided, and ligated, and the ovary attached to the omentum was allowed to slip back into the abdominal cavity. On May 1, 1934, the ovary was removed for examination. Figure 2 shows a typical area from this ovary. There are

many follicles in all stages of development with the primordial phases most numerous. A moderate number of atretic follicles are to the left border of the omentum with black silk. On June 20, 1934, the ovary was removed for histologic examination.

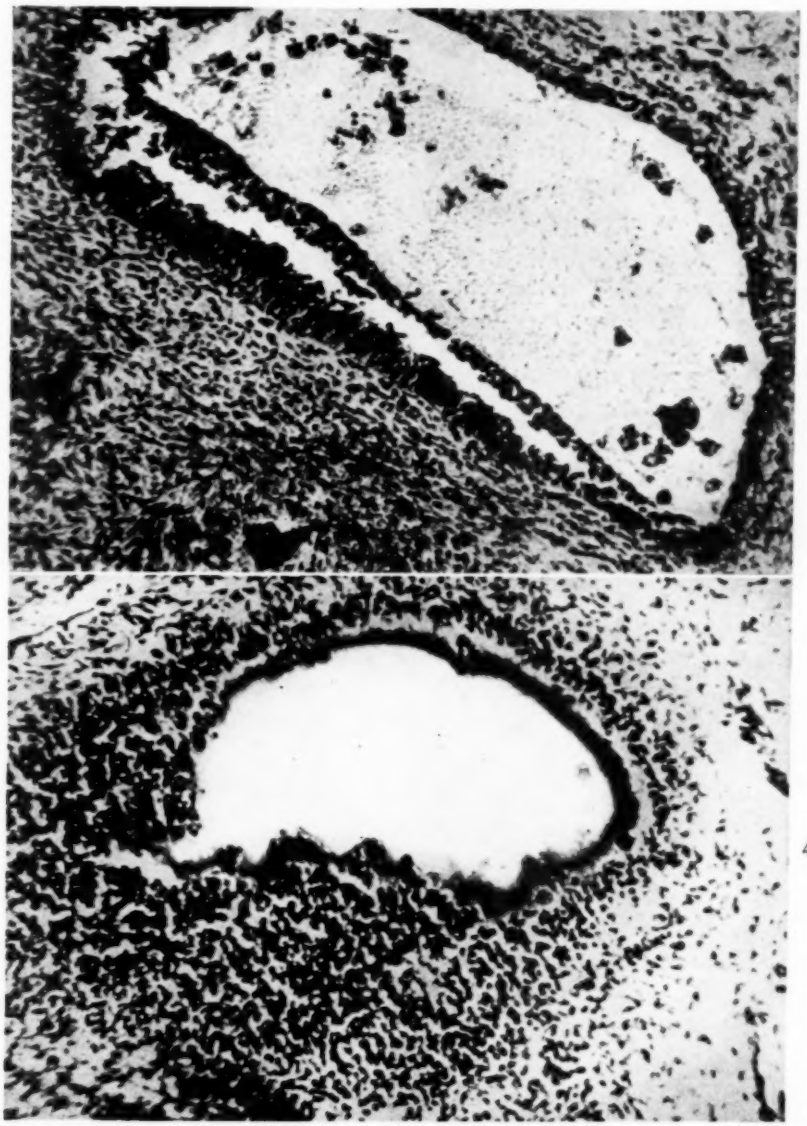

Fig. 3. High power photomicrograph showing detail of follicle with its lining epithelium. Note localized lymphocytic reaction about suture in lower left corner. Ovarian autotransplant forty-five days old.
Fig. 4. Photomicrograph showing a follicle with its lining epithelium. Moderate lymphocytic infiltration below follicle. Ovarian homotransplant forty-five days old.

present. In general, the findings resemble those of experiment 1. The age of the implant is ninety-one days.

EXPERIMENT 3

On May 7, 1934, the left ovary of a dog was exposed in the usual manner. The ovary was freed, bisected and sutured The picture resembled that of experiment 1. Figure 3 is a high power photomicrograph to show the detail of one of the follicles with its lining epithelium. In the lower left corner there is a moderate lymphocytic reaction about the black silk suture. The age of this implant is forty-five days.

EXPERIMENT 4

On May 7, 1934, the right ovary of one dog was bisected and transplanted into the omentum of another dog. A long black silk suture was used for fixing and for future identification. On June 20, 1934, the ovary was removed. Histologically the findings resembled those in experiments 1, 2 and 3. The lymphocytic infiltration is somewhat more pronounced, however, than in the preceding sections. Figure 4 is a photomicrograph showing such an area of lymphocytic infiltration adjoining a follicle which has well preserved epithelium. The age of this implant is forty-five days.

SUMMARY AND CONCLUSIONS

In all the transplanted ovaries examined there was definite evidence of functioning tissue. There was apparently no difference between the findings in the case of the direct autotransplant and the single pedicle autotransplant of dog ovary into the omentum. The lymphocytic infiltration in the autotransplants was minimal and limited to the area surrounding the silk suture. This was interpreted as a foreign body reaction. No giant-cells were seen. There were fewer follicles seen in the homotransplants than in the autotransplants. There also seemed to be somewhat more lymphocytic infiltration and fibrous tissue reaction in the homotransplants than in the autotransplants. The proliferation of the follicular epithelium in the homotransplants was not as marked as in the autotransplants. Leo Loeb says that regenerative processes are usually not very marked in homotransplants of tissue in general. It will be interesting to compare the findings here reported with those observed in transplants of six and nine months' duration. These will be given in a later report and of course will be of much greater practical interest than the ones now reported to those interested in the clinical application of ovarian transplants.

I am indebted to Dr. Emil Novak for his helpful criticism of this paper, and to Mr. Edward Walker of the Surgical Pathology Laboratory, Johns Hopkins University, for the excellent photomicrographs.

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A PLASTIC OPERATION FOR CONGENITAL ABSENCE OF VAGINA

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THERE have been many operative methods used for the construction of a congenital absent vagina and three operative procedures that have been somewhat successful. First, resection of rectum and employment of piece of rectum (Schubert); second, resection of a loop of small bowel (Baldwin); third, transplantation of skin flaps and flaps of labia minora (Graves, Frank, Geist, and Davis and Cron; also Marion Douglass[6]). The operations by Schubert and Baldwin show a high mortality (12.5 to 17.5 per cent). The skin graft and grafts from the labia minora reduce the mortality but are very complicated and the results uncertain. The operation which is herewith presented is quite simple and without any necessary mortality and the end-result is nearest to the normal vagina.

This operation was a three-step procedure over a period of three years, but could easily be completed in six or eight months. This being a charity case, the patient desired to enter the hospital only during the writer's service, which is three months out of each year.

EMBRYOLOGICAL DEVELOPMENT OF THE VAGINA

The generative organs begin to develop at about eight and a half to nine weeks. They come off from the Muellerian ducts and ultimately the Muellerian ducts open into the ventral part of the cloaca between the orfices of the Wolffian ducts and terminate on an elevation named the Muellerian eminence. In the female the Muellerian ducts persist and undergo further development. The portions which lie in the genital cord fuse to form the uterus and vagina. The parts cephalad of

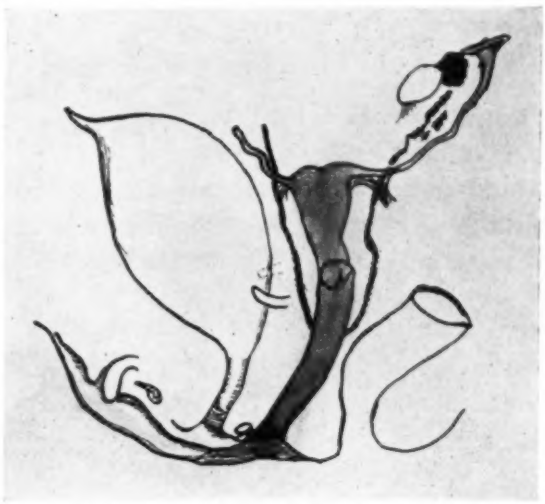

FIG. 1. Diagram of embryological development of female organs at eight to ten weeks.

this cord remain separate and each forms the corresponding Fallopian tube. The fusion of the Muellerian ducts begins in the third month and the septum formed by the fused mesal walls disappears from below upward and thus the cavities of the vagina and uterus are produced. (Fig. 1.) In this case the two sides of the uterus failed to fuse together; consequently, there was no formation of the cavity of the vagina and uterus.

CASE REPORT

The patient, an eighteen year old white girl, entered the Grady Memorial Hospital in June 1932, complaining that she had never menstruated. She was a perfectly normally developed, attractive young woman; breasts

well developed; hair distribution normal; but with no reaction toward the opposite sex.

Inspection of genitalia showed the external

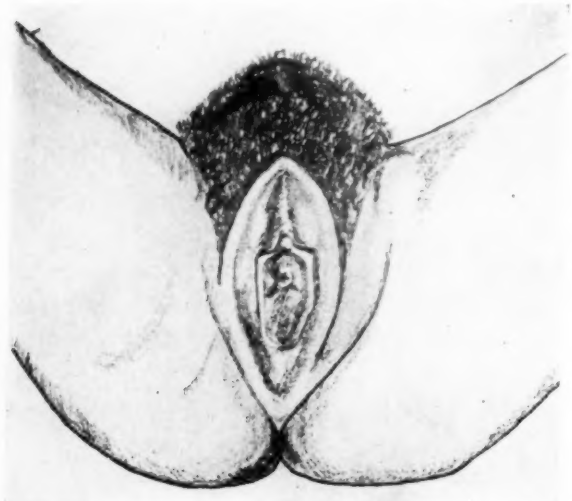

FIG. 2. External generative organs before operation.

genital organs to be perfectly normal except complete absence of vagina. (Fig. 2.) Upon rectoabdominal examination, no uterus could be outlined but both ovaries were palpable and apparently normal in size. A decision was made

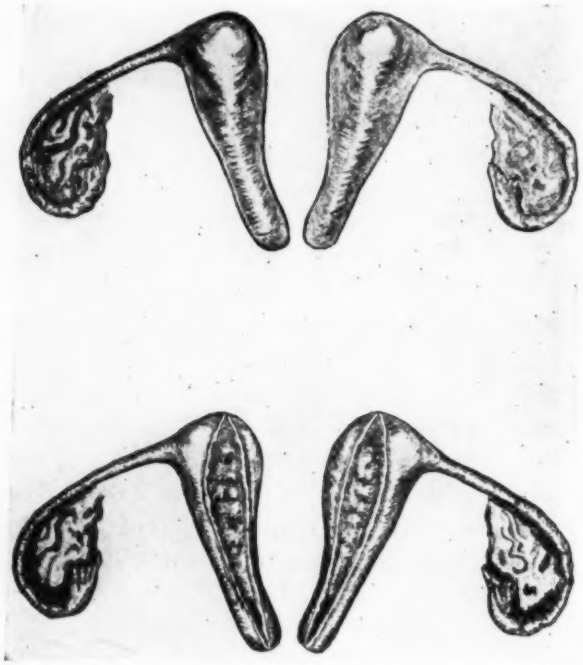

FIG. 3. Diagram showing condition of uterus when abdomen was opened and incisions made preparatory to uniting two sides.

to do an experimental operation to determine if mucous membrane would grow by continuity from the borders of the labia minora.

Operation. Incision was made from just below the urinary meatus down to the posterior fourchette, and by blunt dissection was ex-

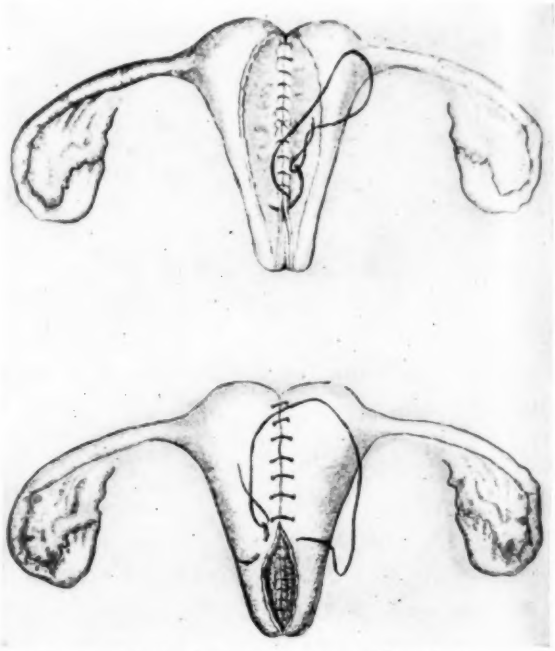

FIG. 4. Method of closing two sides of uterus.

FIG. 5. Actual photograph showing medium bivalve speculum in vagina.

tended 2 inches deep and packed with vaseline gauze. The gauze was removed and repacked every forty-eight hours for one week. At the

end of ten days there was a smooth mucous border on the edges of the incision and at the end of one month the mucous membrane had grown in one inch; the other inch being closed up by scar tissue.

The patient returned to the hospital in December, 1932. On inspection, she had a perfectly normal vaginal opening 1 inch deep. This time incision was extended by blunt dissection 4 inches deep to the pelvic peritoneum; a catheter being in the bladder and an assistant's finger in the rectum as a guide. There was no evidence of any cervix. This cavity was packed with vaseline gauze and treated as before. In one month's time there were 3 inches of mucous membrane, which had the appearance of normal vaginal wall with the usual folds; but the posterior portion was contracted down to a point by scar tissue.

Six months later the scar tissue was dissected out and dilated back as far as the pelvic peritoneum, rectum and bladder would allow. The abdomen was then opened to determine the condition of the internal generative organs. It was found that the two sides of the uterus had failed to unite and each point was attached to the peritoneum with no attachment to the pelvis. A normal tube was attached to the upper prong of each side of the undeveloped uterus with normal ovaries in place. (Fig. 3.)

It was decided to connect the two sides of the uterus and form a cavity. An incision was made down the inner side of each prong of the uterus and extended to the middle. (Fig. 3.) Then the sides of the posterior surface were sewed together, forming half of a cavity. (Fig. 4.) This was packed with rubber tissue. Then the anterior surfaces were sewed together, making a small uterus with an artificial cavity and with tubes going off from each cornea.

A probe was threaded and pushed through the peritoneum at about the place where the cervix should be, into the vaginal cavity. The thread was then attached to the rubber tissue and an opening made large enough so that the rubber tissue could be pulled through the vagina at a later date. The vagina was packed with vaseline gauze and treated as before.

Between the second and third step of the operation, it became evident that the young lady was in love and desired to get married. After she recovered from the operation, the marriage took place. Upon questioning her now, she says that she has a normal desire for coitus and has normal orgasms. There was, however, some contraction and scar tissue in the posterior vault which required dilatation. The vagina is now 4 inches deep, admits the medium size bivalve speculum, and is apparently normal in every respect. (See Fig. 5, actual photograph.) Needless to say, she does not menstruate or can there be a pregnancy due to the lack of uterine mucosa; however, the uterus can be palpated in normal position.

CONCLUSIONS

This operation proves conclusively that mucous membrane will grow by continuity into a cavity where it normally belongs and this seems by far the simplest procedure for the construction of a vagina in cases of congenital absence and the results nearest normal.

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AN OUTLINE OF TREATMENT FOR THE VARIOUS TYPES OF UTERINE ABORTIONS*

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THE treatment of normal uterine abortion is a subject not adequately dealt with in the majority of textbooks on obstetrics and gynecology, yet abortion is one of the very common conditions encountered by obstetricians, gynecologists, general surgeons and practitioners. Nor is it a subject discussed fully enough in our teaching medical schools, even though it is a condition that the young graduated doctor will encounter in his early practice far sooner and more frequently than he will some of the more serious though less common diseases in which he is so adequately trained. According to Taussig[1] between 700,000 to 1,000,000 abortions occur annually in the United States, or about 1 abortion in every 3 deliveries. In addition to the frequent incidence of occurrence of abortion, womankind pays a high mortality rate due to mismanagement of this condition. Septic abortion annually claims 15,000 deaths in this country, and Taussig emphasizes the fact that the death rate from abortion is 7 times greater than that of full term childbirth. The frequent occurrence of this condition and the heavy mortality toll warrant a well-organized and routine method of treatment; such forms the basis of this article.

The treatment of uterine abortion will be considered under 5 headings; (1) threatened, (2) imminent, (3) incomplete, (4) missed, (5) septic.

THREATENED ABORTION

The definition of threatened abortion varies between wide limits. Pelvic cramps, uterine bleeding and dilatation of the cervical os, individually or in any combination, have been described as threatened abortion. It need not be emphasized, however, that many pregnant women encounter pelvic cramps in the early stages of pregnancy entirely unrelated to threatened abortion. Also there are many pregnant women who continue, especially in the early months of pregnancy, their regular monthly flow, yet who follow on to term and are delivered of a normal, healthy baby. It therefore seems that a combination of all three of these signs and symptoms, pelvic cramps, uterine bleeding and a dilated os is the most comprehensive definition of threatened abortion.

The fundamental question to decide is whether an attempt should be made to save cases of threatened abortion, progressed to the stage just defined, or whether the abortion should be immediately completed. Is not spontaneous abortion often one of nature's methods of insuring itself against the propagation of deformed fetuses and monsters? Such a question cannot be answered dogmatically, but must be decided upon by the physician and patient.

Complete rest in bed, with the administration of a sedative, morphine at first, then codein, barbiturates or salicylates later, forms the fundamental treatment of threatened abortion. Packing the vagina is not indicated; in fact, it will often bring on abortion in a pregnancy that otherwise would have continued on to term. Uterine bleeding in any woman, who is put to bed and given a sedative, will usually control itself in its amount of flow equal to or a little less than that of a normal period. Such a flow may continue from three to

[1] Taussig, F. *Am. J. Obst. & Gynec.*, 22: 729, 868, 1931.

* From the Department of Gynecology, Tulane University, School of Medicine.

five days and yet have the pregnancy saved. At the end of this period, if the bleeding shows no indication of slacking, the threatened abortion should be completed. This is probably most readily done by packing the cervical canal and vagina and giving 0.5 c.c. pituitrin twice a day and a course of ergot, a drachm of the fluid extract every four hours for 8 doses. The patient should be placed in the Sims' position, the vagina completely cleansed of any blood clots, and the tip of the iodoform pack introduced into the cervical canal, packing the vagina as tightly as possibly with the remaining gauze. The lithotomy position and bivalve speculum do not offer the necessary facilities for accuracy in packing. The usual outcome, within ten to twenty-four hours, is the onset of pelvic cramps due to uterine contractions, and either the pack is expelled spontaneously, or upon its withdrawal, the uterine contents lie free in the vaginal cavity or can easily be withdrawn with sponge forceps.

If vaginal packing fails to produce the complete abortion, instrumental interference is indicated only if the patient is and has been afebrile from three to five days previously. A febrile condition is not only a contraindication against instrumental uterine interference, but if this procedure is done, very often a fatal outcome or untoward complications result. Uterine manipulation in the presence of fever will be discussed more fully under septic abortions. This interference is not a curettage, but merely a uterine cleansing. The curette is a dangerous instrument in any pregnant uterus due to the ease with which it may penetrate the uterine wall. The sponge forceps or the gloved finger is a much more conservative means of cleansing out an early pregnant uterus. Under gas anesthesia and with the patient in the lithotomy position, 1 c.c. of pituitrin is given to enhance good uterine contraction. Very little dilatation of the cervical canal need be resorted to due to its patency associated with the uterine bleeding. Then a sponge forceps is gently introduced into the uterine cavity and its contents withdrawn as carefully as possible. An iodoform strip is occasionally left in the cavity for twenty-four hours to insure drainage and uterine tone, and upon its removal an occasional bit of uterine content adheres to the strip.

With the advent of endocrinology into general practice, a hormonal treatment for threatened and habitual abortion has been advanced, and in order to understand the rationale of this therapy a short resume of the hormonal control of pregnancy is offered. That the persistence of the corpus luteum influence is essential in the early months of pregnancy seems to be one of the most established facts in the physiology of the reproductive system. Its main hormonal functions are characteristic decidual proliferation of the endometrium for the implantation and nourishment of the ovum, maintenance of pregnancy, and inhibition of ovulation. Survival of the corpus luteum and continued action of its hormone, progestin, is maintained by fertilization of the ovum. Ovarian follicular activity is in abeyance during the early stages of gestation, with follicular hormone excretion in the urine occurring at this time. However, the influence of the corpus luteum hormone, progestin, does not remain on an even level during early pregnancy, but tends to exhibit a rhythmic cadence, somewhat similar to that manifested by it in the normal menstrual cycle. In the early months of gestation, at the would-be menstrual periods, the corpus luteum influence is diminished, relatively or absolutely, and at such periods the occurrence of abortion is most likely to take place, since it is reasonable to assume that insufficient progestin, by failing to maintain the normal decidual growth, might impair the vitality of the ovum. To prevent such a mishap, it would seem logical that the administration of the corpus luteum hormone at such periods, when the menstrual flow is due to appear, would have a prophylactic action in warding off

habitual abortion, and in treating threatened abortion. Progestin is not available commercially, but the luteinizing factor found in pregnancy urine has been administered in a few limited series of cases with encouraging results. Surely when the hormonal products are manufactured in more pure and potent forms, and their actions better understood, the value of hormonal therapy in habitual and threatened abortion will be inestimable.[2]

The treatment of habitual abortion will not be further discussed in this paper since it is so purely an obstetrical condition.

IMMINENT ABORTION

Imminent or inevitable abortion is that stage of uterine abortion in between the threatened and completed types. No matter what may be done in the way of treatment, the abortion is going to occur. Usually fetal and placental parts protrude from a slightly dilated cervix, and the uterine cavity is empty, as demonstrated by a small and well involuted uterus palpable behind the fetal and placental parts. As no further uterine contractions will expel this material, and since infection and necrosis of these parts have generally begun, the immediate removal of the tissue is indicated. The contents can often be withdrawn manually, otherwise the patient should be placed in the Sims' position, the cervix exposed, and the tissue removed from the canal. No subsequent packing is necessary as there is usually very little bleeding, because the tissue, having already separated from the uterine wall, is lying free in the cervical os. However, 1 c.c. pituitrin and a course of ergot should be given to insure continued uterine tone.

Fever in this type of case is usually due to the absorption of toxic products from the infected and necrotic contents in the cervix, and not from any broad ligament or uterine pathology; hence its removal will tend to bring the temperature to normal and there need be no fear of spread of infection because of the lack of uterine involvement.

INCOMPLETE ABORTION

Incomplete abortion is that condition in which the majority of fetal and placental parts have been passed, yet a small remnant of this tissue still remains adherent to the uterine wall, and from this location bleeding occurs. Usually the uterus is increased slightly in size, is subinvoluted, and the cervical os slightly dilated. Such a patient should be watched for a day or so to assure the afebrile state before instrumental uterine interference is undertaken. Then in a manner similar to that described in the completion of threatened abortion, the uterus should be cleaned out with sponge forceps. The initial watching of the patient for a day or so to assure an afebrile state is the most important step in this procedure.

MISSED ABORTION

Missed abortion is that state of pregnancy in which the membranes have ruptured, the fetus dead, yet the uterine contents are unexpelled. Missed abortions generally occur a little later in the pregnant state than the types of abortions already described. The uterus is generally smaller than the suspected month of the pregnancy, and, even though symmetrical, is as hard as a fibroid. Amenorrhea is, of course, present. The treatment should be that indicated for the induction of any normal labor. Instrumental or manual dilatation of the cervix, manual extraction of the fetus and manual removal of the placenta generally result but in one condition, hemorrhage; and this hemorrhage may be so profuse as to become uncontrollable, resulting in death of the patient.

The usual method of inducing labor is to introduce a catheter or two through the cervix into the uterine cavity, and then to pack tightly the cervical canal and vagina. Generally, labor will be induced within twenty-four hours, resulting in the delivery

[2] Witherspoon, J. T. The role of the anterior pituitary luteinizing hormone in threatened abortion. *New Orleans M. & S. J.*, 85: 822, 1932–3.

of the dead fetus and placenta. The postpartal care is similar to that after any delivery.

The point in the treatment of missed abortion which should be emphasized is to induce labor normally and let nature empty the uterus in her own way. Instrumental or manual removal of the uterine contents and placenta is not only contraindicated but is heartily condemned, due to the frequency of subsequent hemorrhage which often follows this manipulation.

SEPTIC ABORTION

Septic abortion is any type of abortion already described that presents a temperature higher than the accepted standard of 100.4°F. Septic abortions are generally induced abortions. This is well illustrated from German statistics in which 80 per cent of women in Berlin who presented this septic condition admitted the induction. It may be well to consider at this point the manner of the introduction of the sepsis. It is not too great a venture to state that a good proportion of women may approach a midwife and have induction performed and "get away with it." If abortion occurs, the outcome may be completely afebrile. It is the woman who does not have the abortion occur after the first induction that pays the price with sepsis. She, already potentially infected, returns to the midwife for the second attempt at induction. This second attempt is the dangerous one, and few women ever escape infection at this time.

Such an unfortunate outcome for the patient is really applicable to the physician who follows, unknowingly or ignorantly, a similar course. If a patient presents herself to a doctor, admitting attempted induction with failure, the only course of treatment is to watch that patient for three to five days without any intrauterine manipulation. If the uterus is entered immediately in an attempt to cleanse it out, the physician is exposing himself to the same unfortunate outcome for his patient as did the midwife. He, too, is entering a potentially infected field, and sepsis will be the usual outcome. If, however, the physician will watch such a patient for five days or so, and if the patient remains afebrile during this period, then, and only then, will it be safe for him to enter the uterus and clean it out. We[3] recently analyzed 200 cases of septic abortion, taking as our criteria of sepsis 101.5°F. of fever, maintained over twenty-four hours. These cases were obtained from the wards of Charity Hospital. One hundred cases were treated by some operative procedure, the minimum of which was the dilatation of the cervix and curettage of the uterus. The mortality in this group was 9 per cent. In a second hundred cases treated only with sedatives, rest and other conservative means, we found a zero per cent mortality. This analysis so substantiated our own clinical experience that at present we are hearty advocates of conservative treatment. Twenty-five to 30 per cent of the patients, who had operative interference done, would have aborted spontaneously on the wards, had operation not been performed, if we can judge by comparison with the conservatively treated group. The duration of fever was three days longer, and likewise the hospital stay was similarly increased in those patients whose uteri were curetted. In contrast, the fall of fever was more rapid in the conservative group. The main outstanding fact of this analysis to us, setting aside temporarily the 9 per cent mortality, was the fact that 64 of the patients whose uteri were curetted were made more septic by the operation as judged by increased febrile reactions, and consequently their convalescence was made more stormy. In 7 women postoperative hemorrhage occurred. In only 13 patients could we find evidence from the histories that operation was a definite benefit to these

[3] Witherspoon, J. T. An analysis of 200 cases of septic abortion treated conservatively. *Am. J. Obst. & Gynec.*, 26: 367, 1933.

women. In addition to the 9 per cent mortality, 11 uteri were perforated during the curettages. Six deaths resulted apparently from these perforations. The contrast of the two types of treatment has been made as strong as possible in order to emphasize more vividly the conservative treatment.

The initial orders are absolute rest under morphine, codein, or any sedative sufficient to obtain the desired result. Feeding of a high caloric, low residue diet is advised; the fluid intake is maintained at 3000 c.c. per day and hypodermoclysis or infusion is resorted to if any nausea or vomiting is present. Multiple small transfusions, 200 to 300 c.c. daily or every two days, are given without hesitancy in desiccation, anemia, or high continued fever. Intravenous antiseptics, foreign proteins and similar therapy have been of little or no aid in this condition.

An initial speculum examination should be made under sterile conditions and any packs, catheters, or foreign bodies immediately removed from the vagina. If remnants of fetal or placental parts are protruding from a dilated cervix, they are gently withdrawn with sponge forceps. However, if no uterine contents are noted, and this is the usual case, no attempt should be made to dilate the cervix or enter the uterine cavity. Sterile pelvic examinations should be made every two or three days to note any evidence of complications outside the uterus, or to ascertain the condition of the exudates in the parametrium or broad ligaments. Should any walling off of these exudates take place or abscess formation occur, the prognosis is much better than in conditions in which the exudates remain generalized. Such abscesses occasionally rupture spontaneously either into the bowel, superficially through the inguinal region or can be drained by posterior colpotomy, as is the usual procedure. No uterine pack, douche, or evacuation should be necessary as these methods of treatment only tend to spread the infection, and what benefits they might accomplish intrinsically are far outweighed by the greater harm they afford in spreading the infection by breaking open large venous sinuses which have already become thrombosed. The chest should be examined daily as septic patients readily tend toward respiratory infections. A blood culture is generally made in the hospital, but its value lies only in diagnostic and prognostic information.

Pituitrin, 0.5 c.c. twice a day, and a drachm of ergot every four hours is the usual procedure in maintaining uterine muscle tone. Under this regime a severe hemorrhage seldom occurs, and it is rarely necessary to pack the vagina; in fact, packing is never resorted to save in rare or extreme cases of severe bleeding, and this only long enough to tide over the acute emergency until the pituitrin and ergot have acted. Even if bleeding continues and the membranes are not completely expelled, a dilatation of the cervix and evacuation of the uterus should not be considered until the temperature has been normal for five days at least, and then only under the most rigid aseptic technique.

It should be emphasized again that the curette is a dangerous instrument in any pregnant uterus, and has no place in the conservative treatment of septic abortion.

PRIMARY CARCINOMA OF THE LUNGS*

COMBINED WITH PULMONARY TUBERCULOSIS

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THE notion of older observers that tuberculosis and cancer are mutually antagonistic has been refuted by recent observations. Not only are the two diseases found in the same individual but their presence has been noted in the same organ, such as the bowel, the genital organs, and the lungs.

In the cases that I have studied, the two conditions were associated in the lungs either accidentally, *per collisionem*, or one played a rôle in the causation of the other.

That one malady may be complicated by another is widely known. Thus diabetes is not infrequently complicated by tuberculosis. The causal relationship between these two diseases is not understood; it is believed that it is probably related to the loss of resistance by the diabetic, thus permitting the infection with Koch's bacillus to take place.

Still more than diabetes, cancer is a debilitating disease and as such it is occasionally complicated by an exacerbation of a pre-existing smoldering tuberculous process. It is interesting, however, that in a few instances when active tuberculosis developed subsequent to carcinoma, the infectious disease was often confined to the lung where the tumor existed while the other lung was spared. It is possible that in these cases the "immured" tubercle bacilli had been released as a result of immunologic as well as mechanical factors.

Whether pulmonary tuberculosis is responsible for the initiation of a primary carcinoma of the lung has been the subject of discussion by investigators who have concurred that in many cases an etiological relationship did exist between the two maladies.

The literature contains articles where the authors reported cases of cancer that had originated in a tuberculous cavity or in a tuberculous scar. Careful study shows that cancer cells found in a tuberculous cavity have not originated *in situ* but in the bronchial wall, ultimately invading the cavity.

The process of "cancerization" of a cavity is analogous to the so-called "epithelialization" of a cavity observed in cases when the infection with Koch's bacillus shows a tendency toward healing.

The wall of certain cavities in the process of healing is made of connective tissue which is not capable of producing epithelial cells. A tuberculous cavity is invariably supplied by a bronchus, the mucosa of which is usually damaged so that its lining no longer contains ciliated columnar and goblet cells but is made up of scattered groups of basal cells. These cells possess a remarkable power for regeneration and usually serve as the sole source for repair of the bronchial mucous membrane. In pathologic processes, these cells grow extensively and invade neighboring tissues and structures. Thus when the disease is active and the cavity contains necrotic (caseated) material, the bronchial basal cells (should they reach the cavity) undergo destruction. But when the cavity is "dry" the cells proliferate along the newly formed connective tissue wall, lining it in a syncytial manner, or they grow in clumps.

An analogous process is observed in instances of cancerization of a "dry"

* Abstract of paper read before the N. Y. Society for Thoracic Surgery.

cavity which is connected with a bronchus whose mucous membrane has become malignant. Cancer cells line the wall of the "dry" cavity in the same manner as they do in instances of invasion of the air vesicles whose walls they often line in an uninterrupted layer.

That a carcinoma will not originate in a "tuberculous scar" which is devoid of epithelial cells is self evident. As in two of the cases that I have studied, the scar was invaded by cancer cells which originated in the mucosa of an adjacent bronchus.

The 13 cases which I have studied occurred in men whose ages varied from forty-five to seventy-one years. In 10 patients, the tumor was on the left side and in 3 on the right side. The upper lobe was involved in 11 and the lower lobe in 2 instances. There were 5 cancers of the small round cell variety, 6 squamous epithelial cancers, one keratinizing epidermoid carcinoma and one adenocarcinoma. The sputum was negative for tubercle bacilli in 10 and positive in 3 patients.

The tuberculous lesion found in the lungs of the patients was of the healing fibrotic type, being to all appearances of long standing. Most of the patients had had a history of cough protracted over a number of years. In 2 cases, there probably occurred a recent dissemination of the bacillary infection (causing a slight exacerbation of the disease) in which the advancing cancer might have played a biologic (lowering of resistance) and a mechanical rôle.

The malignant condition has then developed in cases independently from the tuberculous disease; in others it was engrafted on an ancient fibrotic infection.

In 5 of the 13 cases, a correct diagnosis was made in other institutions but was not suspected by the private physicians who attended the patients for some time prior to hospitalization. Eight of the patients were admitted to the hospital with no diagnosis or with one of tuberculosis of the lungs.

The patients were ill for a considerable length of time and their symptoms and physical signs were compatible with a malignant disease of the lungs. They entered the hospital when the disease was advanced.

The duration of their illness (from the beginning of symptoms) was apparently the same as that observed in patients with pulmonary cancer not complicated by tuberculosis.

In conclusion, it is pertinent to stress that: 1. In any case of chronic pulmonary affection in a person of middle or past middle age, with persistence of symptoms, the possibility of malignant disease of the lungs should be considered. 2. That the presence of pulmonary tuberculosis does not exclude the existence of a malignant process in the same lung.

FATTY INFILTRATION OF URINARY BLADDER

WITH SPONTANEOUS RUPTURE*

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OUR attention was drawn to fatty infiltration (lipomatosis) of the urinary bladder during the histological examination of the urinary bladder of a patient who had died as a result of spontaneous rupture of this organ. The histologic sections revealed a substitution of muscle bundles by fat tissue. Because of the fact that a search through the literature revealed no significant, pertinent data it was decided to study the relation of the perivesical fat tissue to the muscularis of the urinary bladder in a number of normal instances. Since special attention has been paid to this involvement of the urinary bladder by fat tissue, several other observations have been made which also will be reported here.

MATERIALS AND METHODS

In addition to the urinary bladder which had ruptured spontaneously thirty urinary bladders were examined. These were obtained from consecutive autopsies and were from patients who had died from various diseases. Blocks were taken from various regions of the urinary bladder fixed either in Zenker's solution or formalin and embedded in paraffin. Sections were stained with hematoxylin and eosin and according to the Van Gieson's method. When deemed necessary frozen sections were cut and stained for fat with Sudan III.

RESULTS

Normally the urinary bladder is surrounded by fat situated just beneath the peritoneal covering. Anteriorly, adjacent to the perivesical spaces of Retzius a considerable amount of fat tissue is also present. The amount of fat varies, being, in general, proportional to the amount of adipose tissue throughout the body. Usually more fat is seen grossly in the posterior wall of the bladder and in the fundic region than in the anterior wall. Histologically the fat tissue is distinctly separated from the peritoneal covering and from the musculature. The line of demarcation between the fat tissue and the muscularis is very sharp.

In three instances there was an extension of the perivesical fat tissue into the muscularis. In one of these bladders the fat within the musculature was recognized grossly. This was in the bladder which had ruptured spontaneously. In the two other instances the extension of fat tissue into the muscularis was discovered in the microscopic sections. Only one patient was obese. The other two seemed to have a normal amount of adipose tissue.

Histological examination of some sections revealed that fat had invaded the peripheral portions of the musculature replacing only the outer layer. In other sections fat tissue was found in the interstitial spaces of the muscularis at a considerable distance from the sub-peritoneal fat. The fat here seemed to follow the smaller blood vessels. In other instances large portions of muscle fibers were replaced by fat which had extended as far as the submucosa. The submucosa in places was partially replaced by fat. This extensive replacement was most marked in the bladder which had ruptured spontaneously. Often islands of muscles

* From the Departments of Pathology and Urology, Michael Reese Hospital. Aided by a grant from the Otto Baer Fund.

were seen which were completely surrounded by fat. Some of the muscle fibers were distinctly atrophic, their cytoplasm was granular and nuclei were hardly visible. Very occasionally only remnants of muscle fibers were seen. In a few histological fields the entire wall of the urinary bladder consisted of fat.

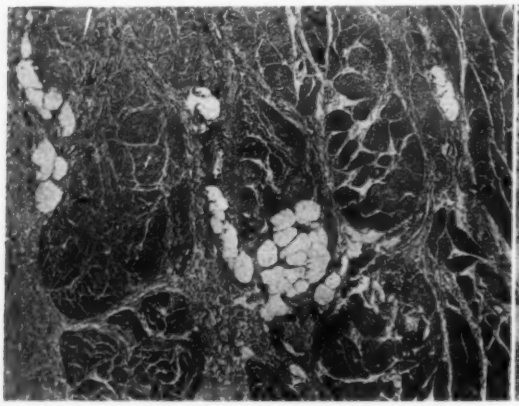

Fig. 1. Early fatty infiltration of urinary bladder. Note accumulation of fat cells in musculature. Hematoxylin-eosin preparation × 40.

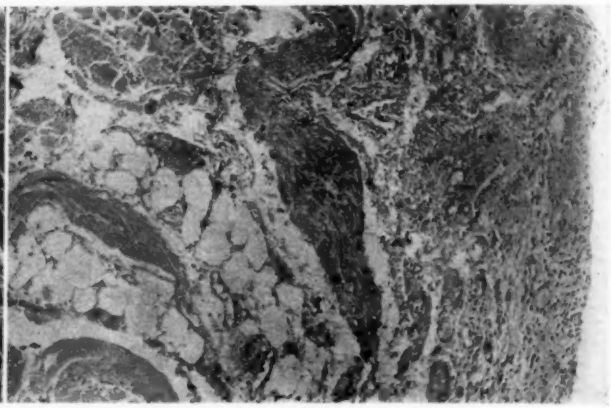

Fig. 2. More severe fatty infiltration of urinary bladder. Fat is present in muscularis close to submucosa. Hematoxylin-eosin preparation × 50.

It may be of interest to note that in two instances other changes besides fatty infiltration were found. In one a chronic cystitis was present, in the other a diffusely infiltrating primary carcinoma. As previously mentioned the fatty infiltration in these two instances was an incidental finding. Only in the case of spontaneous rupture was fatty infiltration the sole abnormality encountered in the urinary bladder.

Since little is known of fatty infiltration of the urinary bladder it may be of interest to compare this condition with fatty infiltration of another muscular organ, the heart, which often is the seat of fatty infiltration. By fatty infiltration of the heart (myocardium) is meant the formation of an abundance of sub-epicardial fat which extends into the myocardium and occupies the usual position of muscle fibers. By analogy fatty infiltration of the urinary bladder may be defined as the formation of an abundance of sub-peritoneal fat which extends into the musculature of the urinary bladder and occupies the usual position of the muscle fibers. Because fatty infiltration was found in two instances without causing any clinical symptoms we believe that this condition may be present anatomically but be silent clinically. The same is true of fatty infiltration of the myocardium, which condition is often found at autopsy in the absence of a history of heart failure (Saphir and Corrigan). Apparently the myocardium and the urinary bladder which are the seats of fatty infiltration may carry on their functions as well as the normal heart and the normal urinary bladder. But these organs when infiltrated with fat have very little reserve power. The heart may fail unexpectedly if its work is suddenly increased. Extremely rarely it may rupture. The urinary bladder may rupture as the result of insignificant causes. One case in this series is characteristic in this respect and shall be given in more detail.

Clinical History: A white male, fifty years old, had been at a party at which he had consumed a large quantity of beer and wine, becoming quite intoxicated. He was suddenly seized with severe abdominal pain, associated with nausea and vomiting. Since the onset of the pain he had been unable to urinate. Members of his family, who had been with him before the onset of the pain and since then, denied any injury or fall of the patient. The patient was seen by the family doctor the next day. Upon catheterization a bloody urine was

obtained. The patient was brought to the hospital about twenty-four hours after the onset of the pain.

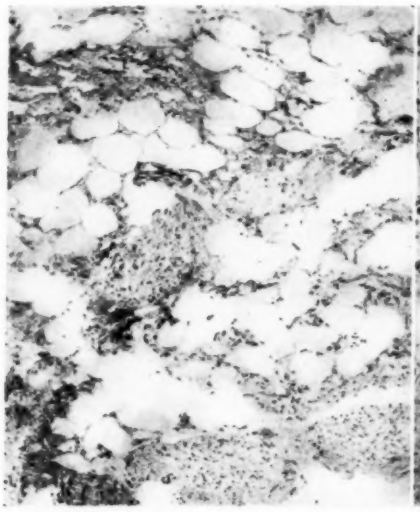

Fig. 3. Almost complete replacement of muscularis by fat. (Case of ruptured bladder.) Hematoxylin-eosin preparation × 50.

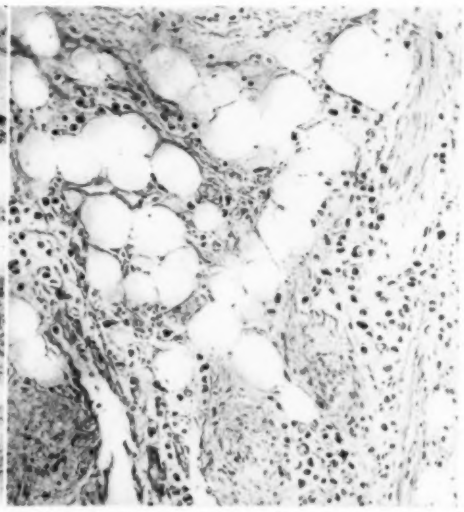

Fig. 4. Fatty infiltration of urinary bladder combined with diffusely infiltrating carcinoma simplex. v. Gieson stain × 100.

On admission the patient appeared acutely ill. The pulse rate was 100 per minute. Temperature was 100.2°F. rectally. The abdomen was markedly distended, tympanitic anteriorly and dull in both flanks. The area of dullness shifted with change in position. The rectal examination was negative. There was no sign of any bladder distention. Catheterization was easily carried out. At first no urine was obtained by inserting the catheter to the usual depth, but by pushing the catheter to its full length into the bladder 1500 c.c. of bloody urine were obtained. Because of these findings the diagnosis of intraperitoneal rupture of the bladder was made and the patient was immediately operated on.

A mid-line sub-umbilical incision was made and the peritoneum opened. A large quantity of bloody purulent fluid escaped. The bladder was contracted and a large tear 3.5 cms. long was found in the vertex. The bladder was palpated, but no pathological changes could be found. The tear was sutured, the peritoneal cavity drained and a retention catheter fastened into the urethra.

The patient stood the operation well, but shortly thereafter the temperature rose to 104°F. and the pulse frequency was 120 per minute. There was complete anuria and the patient died thirty-six hours following operation.

At autopsy about 200 c.c. of a turbid fluid with pus and flakes of fibrin were present in the peritoneal cavity. The urinary bladder contained a small quantity of a turbid yellow fluid. In the posterior wall a recently sutured laceration, 3.5 cm. in length extended in a roughly horizontal direction over an area located about 3 cm. above the uretheral orifices. The laceration reached the fundus of the bladder penetrating the entire wall. The sutures were intact. Immediately surrounding the site of laceration the mucosa was hemorrhagically discolored and covered with fibrinous excrescences. The fatty tissue surrounding the bladder was increased and fat was seen grossly in the musculature. There was no obstruction at the neck of the bladder or in the urethra.

Histological examination revealed the replacement of the muscularis by much fatty tissue which in some fields could be demonstrated throughout the entire wall of the urinary bladder. In one area, the site of the rupture, necrotic tissue, many red blood corpuscles and polymorphonuclear leucocytes were recognized. Suture material was also seen in this region.

This case we believe shows the extreme degree of fatty infiltration of the urinary bladder. While the patient was intoxicated he suddenly experienced pain in the bladder region. The bladder apparently was markedly distended. A normal urinary bladder would have sustained this strain. However

a bladder in which the muscle fibers were largely replaced by fat was unable to withstand the large intravesicular pressure

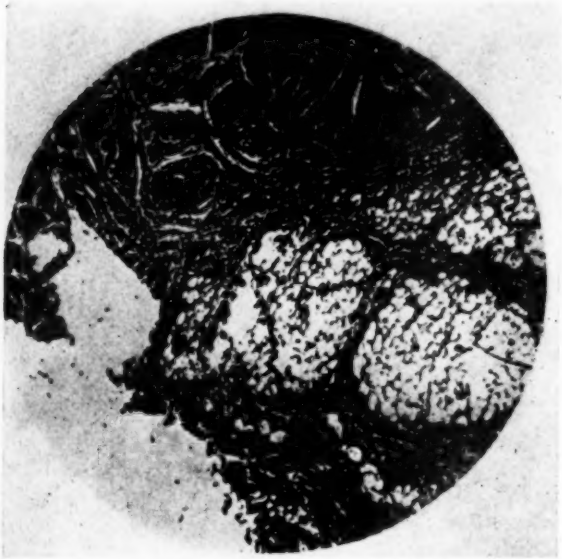

Fig. 5. Picture taken from Bitschai's article and called "place of rupture of urinary bladder." Note presence of fat.

and ruptured.

It is of interest to note that the rupture occurred while the patient was intoxicated. This is a frequently encountered coincidence. Campbell mentioned that one-third of 55 patients suffered ruptures during the state of acute alcoholism.

It is a well-known fact, which has been recently again emphasized by Neugebauer, that rupture of the mucosa and submucosa is dangerous in that the rupture may later involve the muscularis and result in a perforation. Fatty infiltration involves primarily the muscularis, but spares the mucosa and submucosa in its early stages. After the rupture of the mucosa and submucosa has occurred the fat tissue may subsequently give way thus causing the complete rupture. If a rupture of the mucosa and submucosa extends into the muscularis, the latter should be examined for fat. A careful histologic examination of the urinary bladder in such an instance may reveal a fatty infiltration and possibly explain the extension of the rupture into the muscularis.

As was mentioned before we were unable to find significant references in the literature on fatty infiltration of the urinary bladder. A few pertinent data which we were able to accumulate may be given. Koelliker in his comprehensive text on normal histology remarked that the subperitoneal fat tissue here and there follows the course of the nerves and blood vessels and often extends deeply into the muscularis and occasionally also into the submucosa. Sisk and Wear stated as follows:

"The bladder wall (in instances of spontaneous ruptures) may be the seat of carcinoma, tuberculous and syphilitic ulcers, lipomatosis, degeneration of the musculature and old scars." These authors mentioned lipomatosis but gave no details. Naumann described a case of rupture of the urinary bladder and mentioned that the patient had a fatty heart. The amount of fat around the urinary bladder or within the urinary bladder was not mentioned. Campbell also mentioned degeneration as a co-existing lesion in the urinary bladder in instances of ruptures. However, he neither mentioned fatty degeneration nor fatty infiltration. Fahr, quoting Gudynski, enumerated fatty degeneration among the causes of rupture of the urinary bladder. It may be mentioned in this connection that fatty infiltration and fatty degeneration have nothing in common. Fatty degeneration is a deteriorative process in the course of which fat becomes apparent in the cells, while fatty infiltration affects cells which normally contain fat, and represents an alteration simply of the normal fat depots and fat transport (Karsner).

Dittrich mentioned lipomatosis among diseases of the urinary bladder which may lead to rupture. Only two references were found in which fatty infiltration of the urinary bladder was actually described. Herting reported 3 instances of ruptured bladder. In all three a fatty infiltration was noted. The patients died of ruptured bladder. The author, however, believed that not the fatty infiltration but

a degenerative process of the muscularis which was also present had caused the rupture. Because the 3 patients were inmates of an insane asylum this article was published in a journal of psychiatry and escaped the notice of urologists, surgeons and pathologists. Hedrén described the only case of fatty infiltration of the urinary bladder in which the rupture of the bladder was attributed solely to the replacement of muscle fiber by fat.

Fatty infiltration of the urinary bladder probably would be found more often in instances of rupture, if this condition were searched for and recognized and if it were accepted as a predisposing factor in spontaneous rupture of this organ. This became evident to us after studying Bitschai's case report of spontaneous rupture of the urinary bladder. This bladder revealed an epithelioma. The author, however, stressed that the site of rupture showed no tumor. There was no mention made of the presence of fat in the urinary bladder. One of his illustrations, however, shows a typical fatty infiltration of the muscularis, and is here reproduced.

We are well aware of the fact that fatty infiltration of the urinary bladder per se is probably not the cause of spontaneous rupture. But from our study we believe, that like the fatty infiltration of the myocardium, it reduces the muscular resistance of the urinary bladder to such a degree that apparent insignificant causes such as marked distention of the bladder may result in rupture.

SUMMARY AND CONCLUSIONS

Fatty infiltration of the urinary bladder is characterized by the formation of an abundance of subperitoneal fat which extends into the musculature of the urinary bladder, and occupies the usual position of the muscle fibers. Apparently the urinary bladder which is the seat of fatty infiltration may carry on its function as well as the normal urinary bladder but has little reserve power. The urinary bladder which is infiltrated by fat may rupture as a result of apparent insignificant causes. Fatty infiltration of the urinary bladder was found twice among thirty bladders examined. In one other instance the fatty infiltration was so severe as to have led to rupture.

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FRACTURES OF THE BONES OF THE FOREARM

A DISCUSSION OF NON-OPERATIVE TREATMENT AND REPORT OF 150 CONSECUTIVE RECENT CASES

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THE non-operative treatment of fractures of the bones of the forearm is better understood when one recalls the skeletal anatomy and the intricate muscle mechanisms involved in fractures of the radius and ulna. I shall confine my discussion of the treatment and principles involved to the cases of my series in which the patient was under sixteen years of age.

ANATOMICAL CONSIDERATION

The ulna is directly continuous with the humerus. It is a fixed hinged joint and is concerned only with flexion and extension. The ulna does not take part in inward rotation (pronation) and outward rotation (supination) of the forearm. The radius is continuous with the hand. At the upper end and the lower end, the radius rests in the ulna. The radius alone moves, revolving about with the ulna as an axis, thus allowing pronation and supination. The radius is concerned with the motion of the hand, the ulna with that of the arm. The interosseous space is at its maximum in supination, while in full pronation the radius nearly contacts the ulna. The head of the radius rotates in the orbicular ligament; the lower end of the radius revolves around the head of the ulna and rests in the interarticular triangular cartilage. The range of motion is 140°–160°.

The anatomy of the inferior radio-ulnar articulation is important when one considers the intricate mechanism of the forearm. The term, "dislocation of the head of the ulna" is a misnomer. The ulna is fixed. It does not dislocate. It is the lower extremity of the radius which undergoes the displacement and it carries with it the carpus and the hand. The triangular disc, fixed by the apex to the joint at the base of the styloid process of the ulna and by its base to the distal ridge of the ulnar notch of the radius, is the most important ligament in the activities concerned with rotation at the radio-ulnar joint. This joint is at its weakest from the point of view of muscle control. The superior radio-ulnar joint plays a part in fractures, with displacement, of the upper half of the ulna. If the displacement is marked, there must result a fracture of the head of the radius or a displacement of the upper end.

The muscles of the forearm divide themselves into three groups (Davis):

1. Those which extend the fingers.
2. Those which flex and extend the wrist.
3. Those which pronate and supinate the radius and the hand.

Radius is pronated by

(1) M. pronator quadratus.
(2) M. pronator radii teres.

Radius is supinated by

(3) M. supinator brevis.
(4) M. supinator longus (Brachio-radialis).
(5) M. biceps.

Muscle action plays an important part in displacement of the fractures of the radius and ulna. There are four types:

1. There may be simple over-lapping. (Fig. 3.)
2. There may be obliteration of the interosseous space because the pronator quadratus and teres both pass from the ulna to the radius, the one at the lower end,

the other at the upper portion of the forearm. When they contract they tend to draw the bones together. (Fig. 10.) The

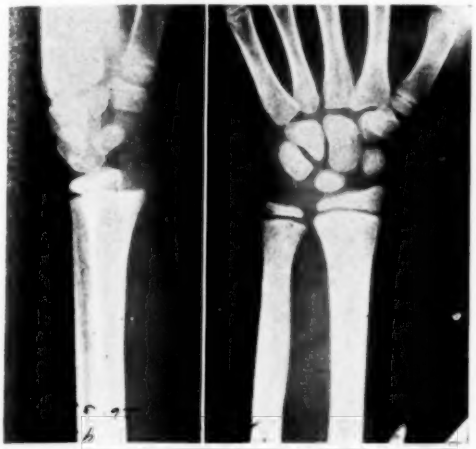

Fig. 1. Epiphyseal separation of distal end of radius dorsally. Before reduction.

brachioradialis contracts and tends to tilt the upper end of the lower fragment toward the ulnar side.

Fig. 2. Fig. 3.

Fig. 2. Epiphyseal separation of distal end of radius dorsally. After reduction. Note hand is dressed in palmar flexion.

Fig. 3. Fracture of radius and ulna with overlapping.

3. Fragments may be rotated on one another in the direction of pronation and supination. When the fractures occur above the insertion of the pronator radii teres the upper fragment is rotated outwardly by the biceps and supinator brevis. There are no muscles to oppose them. When the bones are broken below the middle of the

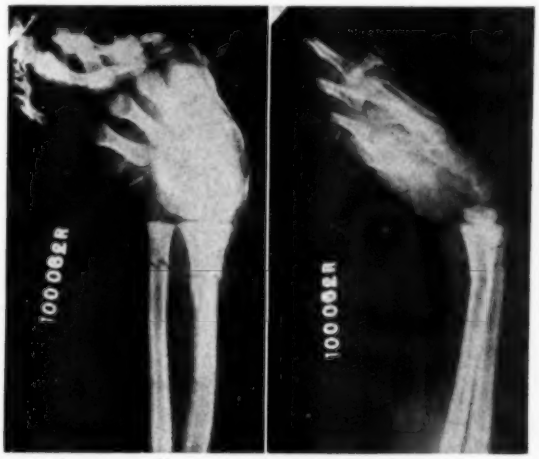

Fig. 4. Case 149 illustrated in Figure 3. After reduction.

forearm, the pronator radii teres remains attached to the upper fragment. Considering the supinating action of the biceps and

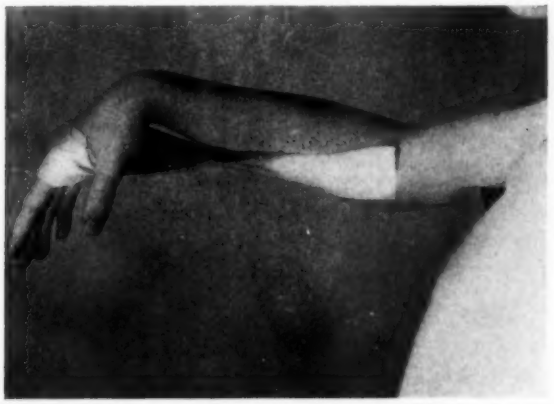

Fig. 5. Method used to hold reduced epiphyseal separation of distal end of radius dorsally and fracture of bones within 1½ inches of joint. Note application of adhesive plaster.

the supinator brevis, therefore, the fracture is treated midway between supination and pronation (thumb up).

4. Angular deformity or simple bending at the site of fracture. (Fig. 14.)

MATERIAL

I have divided this non-operative series of 150 fractures of the forearm, which have been treated by me during the last seventeen months, into adults and those sixteen

years or younger. There were 49 adults and 101 children (67 per cent).

Complete fractures of the distal ends of the radius and ulna in children and adolescence are a distinct entity and must not be confused with the so-called Colles' fractures.

Fig. 6. A muslin bandage covers adhesive plaster as illustrated in Figure 5.

Table 1

Site of Fracture	Adults	Children
Colles' fracture	25	0
Epiphyseal separation of distal end of radius dorsally	2	10
Radius below pronator radii teres	5	30
Ulna below pronator radii teres	3	5
Radius and ulna below pronator radii teres	0	28
Radius above pronator radii teres	0	5
Ulna above pronator radii teres	3	8
Radius and ulna above pronator radii teres	3	15
Head of radius	7	0
Ulna with dislocation of head of radius	1	0
Total	49	101

The oldest patient with epiphyseal separation was twenty years, the youngest ten years. Ten of the patients were under sixteen years. In the total number of children 73.7 per cent of the fractures were in the lower third. The left forearm was fractured twice as often as the right. The radius usually fractured higher than the ulna.

TREATMENT

The treatment of fractures of the bones of the forearm, in children and adolescents, may be classified according to the relationship of the fractures to the insertion of the pronator radii teres and the treatment of greenstick fractures:

I. Epiphyseal separation of the distal end of the radius dorsally and fractures of the bones within 1½ inches of the joint

Fig. 7. Fig. 8.

Fig. 7. Traction cast in use, not new, but serviceable in nonoperative treatment of fractures of bones of forearm.

Fig. 8. Plaster-of-Paris cast, with forearm in full supination.

line are best dressed, after reduction, in palmar flexion (complete pronation). (Figs. 1–6.) No splint or plaster-of-Paris is needed, just adhesive and a muslin bandage. The reduction of the separated epiphysis is not difficult if the case is recent, but if it is two weeks after injury, one must operate. Complete fractures of the radius and ulna at the lower end are best reduced under the fluoroscope and then are dressed in palmar flexion as described. After two weeks of this treatment, the forearm is dressed in a straight position with a muslin bandage and the patient continues to carry the forearm in a sling.

II. Complete fractures of the radius and ulna below the pronator radii teres and above the pronator quadratus.

A. The transverse fractures are reduced under the fluoroscope and held with a well-padded right angle splint or a plaster-of-Paris cast, the joint above and below the fracture being fixed. The forearm is held in the semi-pronated position (thumb up) because of the mechanics of muscle pull as already described.

B. The oblique fractures are held in reduction by the traction cast. (Fig. 7.) The traction and counter-traction will overcome the muscle pull on the fragments and the semi-pronated position will accom-

plish the reduction. After two or three weeks of traction the union is sufficiently solid to allow the molding of the fragments by means of felt pads. Any forearm splint or a wedge cast may be used.

B. Fractures below the pronator radii

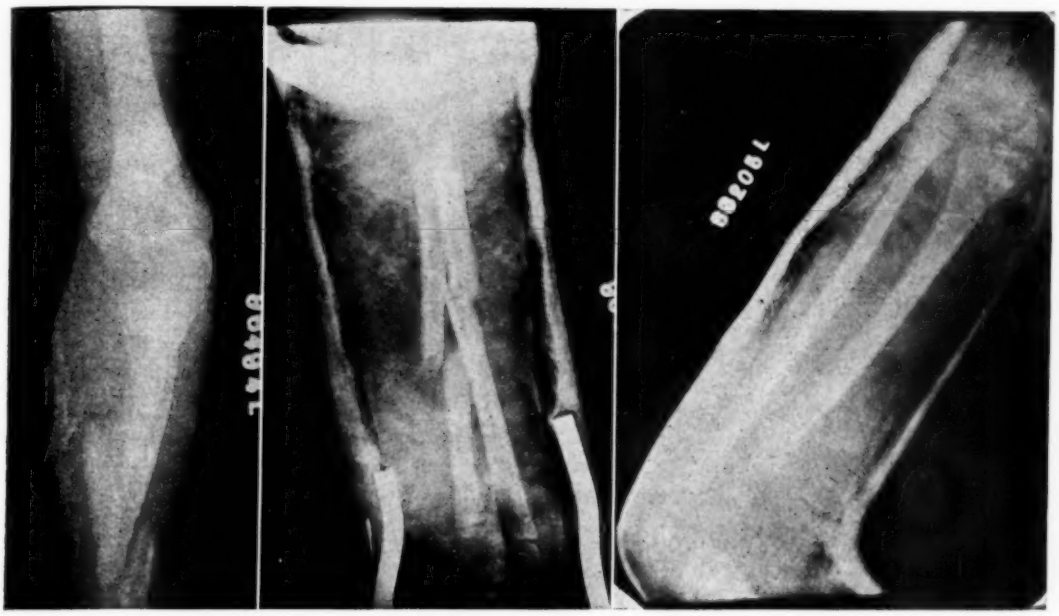

Fig. 9. Fig. 10. Fig. 11.

Fig. 9. Case No. 24. Complete oblique fractures of radius and ulna above pronator radii teres.
Fig. 10. Case No. 24. After application of traction cast.
Fig. 11. Case No. 24. Six weeks later.

and the application of a snugly fitting cast.

III. Complete fractures of the radius and ulna above the pronator radii teres. It is noted that the proximal fragment of the radius is always in supination.

A. Transverse fractures are reduced under the fluoroscope and held by means of a cast with the forearm in full supination and the elbow at 80°. (Fig. 8.) However, if placing the forearm in supination causes a great increase in bony displacement, then that position which will assist in maintaining the reduction should be used. I do not recommend the use of the wooden, internal right angle splint, as I have seen several cases of ischemic paralysis follow their use.

B. The oblique fractures are held reduced by means of the traction cast (Fig. 7), the forearm being fully supinated. (Also see Figs. 9–11.)

IV. Incomplete or greenstick fractures of the radius and ulna.

A. Fractures within 1½ inches of the wrist joint are molded and held in position teres and above the pronator quadratus are treated by means of a well-padded wedge cast. The forearm is held in the semi-pronated position (thumb up).

C. Fractures above the pronator radii teres are held in good alignment by means of a well-padded wedge cast with the forearm fully supinated. The elbow is held at 80°.

V. Fracture of the ulna.

The position of the fracture depends on direct violence. The distal fragment is most often displaced to the radial side because of the pull of the pronator quadratus muscle. The upper fragment, articulating with the humerus by a pure hinged joint, cannot be displaced itself, but the radius and hand can move bodily toward the ulna.

Fractures of the ulna are treated with the forearm in full supination.

After-treatment: Start at once to move the fingers and shoulder. All casts are to be split. (Figs. 7 and 8.)

Board splints should be wider than the forearm, thus avoiding squeezing together of the bony fragments and constriction of

Fig. 12. Wedge cast. Felt pads are applied after first covering arm with sheet cotton.

the circulation. Bowing of the fractured fragments is best avoided by having a snugly fitting arm cast about the elbow joint. If the board splint is used, multiple strips of zinc oxide adhesive are fastened to the arm above and below the elbow. The ends of the adhesive strips should not overlap. Slings should be adjusted so as to lift high the forearm and shoulder girdle. Those patients wearing the traction cast are observed frequently for early signs of ischemic paralysis.

Comment: None of the 101 children was operated on. However, there were 3 cases in which the radius was compounded, but there was no internal fixation used when the bone was replaced. None of the cases was treated by suspension traction. Ten per cent of the patients were hospitalized. There were two refractures. There were no permanent nerve injuries or ischemic paralysis.

SUMMARY AND CONCLUSIONS

1. Non-operative, manipulative mechanical treatment of fractures of the radius and ulna in children and adolescents ends in a good functional result.

2. The knowledge of the intricate muscle mechanism in fractures of the radius and ulna will assist in the non-operative treat-

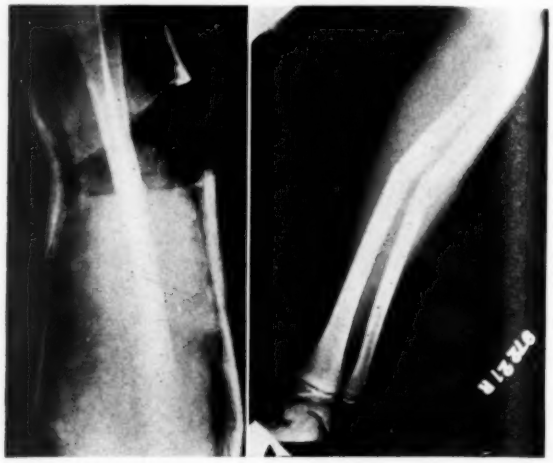

Fig. 13. Fig. 14.

Fig. 13. Wedge cast used to straighten out a greenstick fracture.

Fig. 14. Angular deformity or simple bending at site of fractures.

ment of these fractures.

3. Epiphyseal separation of the distal end of the radius dorsally and fractures of the bones within 1½ inches of the joint line are best dressed, after reduction, in palmar flexion. Roughly, fractures of the upper third of the forearm are dressed in supination while fractures of the lower two-thirds are dressed in semi-pronation.

4. A perfect anatomical result, although desired, is not essential for a good functional result.

5. The real value of the study of these cases is, we conclude, that operative interference is rarely justified in patients under sixteen years.

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FORENSIC MEDICINE

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CONCUSSION OF THE BRAIN*

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CONCUSSION of the brain is often produced by a severe blow on the head and is marked by unconsciousness, shallow respiration, slow pulse, cold skin and pallor, often followed by partial stupor, vomiting and severe headache. The respiration may occasionally be so shallow that the patient gives the appearance of being dead. In most cases, however, consciousness is regained within a few minutes and these are therefore called "slight concussion." When consciousness is not regained for several days the case is described a "serious concussion." Occasionally one encounters a severe case in which death occurs almost instantaneously. Fortunately such cases are very rare. Nevertheless, such cases are the main concern of this discussion inasmuch as several of these are reputed to have shown entirely negative findings at autopsy. These so-to-speak wholly negative pathological cases have greatly contributed to shroud the true etiology of *commotio cerebri* in mystery. This lack of clear understanding is reflected in the very term "concussion of the brain." A disease is usually designated according to the pathological lesions generally encountered in affected organs. Hence the names encephalitis, meningitis or acute anterior poliomyelitis are employed to describe the inflammatory condition in the areas affected. The name "concussion of the brain," however, does not embrace the pathological changes in the brain, but only describes the cause of the disease condition.

Medical historians trace the disease back to the schools of Hippocrates and Galen. The French physician Boirel in 1677 was the first to employ the term "commotio" with reference to cranial injuries, much in the same sense as it is used at the present time. Thus he distinguished concussion of the brain from other conditions which might likewise result from injuries to the head, such as contusion and compression of the brain. But because of the wholly negative pathological findings at autopsy such a case of fatal head injury was called "pure concussion" and a classical example of this entity is described by Littré in 1705. A young prisoner about to be executed, committed suicide in jail by running and dashing the head against the wall. Death took place instantly without the prisoner uttering a sound. The autopsy revealed a brain diminished in volume and of firmer consistency than normal. This picture differs decidedly from the usual autopsy findings in persons dead from head injuries who presented the clinical picture of "*commotio cerebri*" before death. One finds as a rule in such cases hemorrhages in the cerebral cortex and more or less contusion of the surface of the brain, besides the fracture of the skull.

* Address delivered before the Norwegian Academy of Science.

In considering the question of the etiology of the typical case of concussion of the brain, one may choose between two interpretations and these depend on the attitude one adopts towards the autopsy-findings in persons dead from head injuries and who while alive presented the typical clinical picture of a "pure concussion." If one does not accept the autopsy findings, in the belief that several important things were overlooked, such as small superficial contusions, one might assume that these slight lesions are the probable cause of the concussion phenomenon. If, on the other hand, one accepts the accounts of the "pure concussion," one would be forced to consider that it was neither the actual contusion of the surface of the brain nor the hemorrhages connected therewith which were the cause of the condition, as far as the ordinary autopsies are concerned. It is probable that two coordinate processes take place in the injured brain, one of which gives rise to the ordinary symptoms of concussion, while the other calls forth only the usual symptoms of contusion of the brain, which include various kinds of paralysis or deficiency phenomena. Under ordinary conditions, the contusion of the surface of the brain gives at first rise only to slight symptoms or to none at all, the picture being completely dominated by the concussion. The signs of "concussion" gradually disappear after a few days, while the signs of contusion of the brain surface increase in evidence.

The conception formed in the course of time as to the nature of the slight concussions has its origin in the belief in the correctness of the wholly negative autopsy findings in the "pure concussion" case, such as when the patient is allowed to return home shortly after admission to the emergency clinic. The only plausible explanation of the condition in such cases is that the concussion is a disturbance of purely functional nature and without any visible organic lesions. This view agrees with the hypothesis that the trauma which brings about the functional disturbance is due to the oscillations within the cranium. This leads to the so-called "molecular" oscillations supposed to produce the invisible "molecular changes" in the nerve tissue as being the cause of the disorder. This hypothesis is certainly due to a deficient knowledge of the true pathology of concussions. Even as recently as in 1921, Bresslauer and Rahm discussed in *Bruns Beiträge* whether Newton's laws of mechanics or Einstein's theory of relativity should be used as basis for the consideration of the nature of concussion of the brain. The latter view is held by Rahm and the terms "induced gravitation field," "induction of the field of gravity," "translation movement," etc., are due to him. The actual text is as follows: "Dass die Gravitationsfeldwirkung direkt auf die Gangliezellen Ursach der Commotionssymptomen ist, dass glaube ich auf Grund allgemeiner klinischer Erfahrungen annehmen zu dürfen." (That the action of the gravitation field direct upon the glanglion cells is the cause of the concussion symptoms I believe admissible on the basis of clinical experience.) Amid this confusion of verbosity one is reminded of Rebecca's remark in Ibsen's "Rosmersholm" about "intricate and misleading paths."

Towards the end of the eighteenth century, experimental pathology was greatly spurred on in France by the prizes offered by L'Académie de Chirurgie for problems dealing with head injuries and in particular with indirect fractures of the cranium. At that time the trauma was supposed to bring about oscillations of the cranium and to fracture the skull at its weakest point. The oscillations were furthermore supposed to be transmitted through the cerebrospinal fluid to the brain. In this manner the French investigators were led to believe in the molecular changes already mentioned.

In the course of time the so-called "pure concussions," including Littré's own case, became subjected to severe criticism and

most of the cases were discarded from this group inasmuch as their supposed "pureness" was considered to be due to imperfect investigation. But the belief prevailed stubbornly that concussion of the brain had no anatomical basis. Thus Fano states that his belief was mainly supported by the fact that the Frenchman Sabatier in 1799 described a case analogous to that of Littré. According to Fano, this brought about a revival of the hypothesis of "pure concussion" and he adds that during the first three decades of the nineteenth century this notion was generally accepted "sur la parole et sur la foi des maîtres." In the meantime those investigators who believed that concussion of the brain had an actual anatomical basis they could not agree as to its locality in the brain. And in view of the constant and well-defined symptom complex characteristic of the concussion of the brain one should reasonably be expected to find a constant site for these anatomical changes. The delay in discovering the actual site of the lesions in concussion of the brain is best understood in the light of the historical development of the study of the pathological anatomy of the traumatic brain lesions. In his lectures in 1855-56 Rudolph Virchow contended that pathological anatomy as a science dated only from the last decades of the previous century, for even as late as in the seventeenth century and certainly in the latter part of the sixteenth century "every physician knew everything and every professor almost more than everything." If one desires to ascertain what a surgeon in Napoleon's time knew about the pathological anatomy of the traumatic brain lesions, one should read Dupuytren's "Leçons orales de clinique," although they were published years afterwards. Dupuytren was no doubt one of the foremost surgeons in his time, greatly interested in pathological anatomy and having had a great deal of battlefield experience. His lectures, however, present nothing of importance on concussion of the brain except for a description of the surface of the brain after contusions, as well as a description of the effect of "*contre-coup*," which Fallopius had already described as early as in 1550. Traumatic hemorrhages in the interior of the brain, especially in the ventricular walls, are not mentioned either by Dupuytren or Rokitansky who published the first textbooks on pathological anatomy in the modern sense of the word. This last-mentioned book appeared between 1842 and 1846, while the volume dealing with the central nervous system was published in 1844. Neither does one find any mention of ventricular hemorrhages following concussion of the brain in the textbook or atlas written by Cruveilhier.

In his discussion of concussion of the brain, Rokitansky refers to the occurrence of the "pure concussion," of which Littré's case is a model example. Thus one finds one author after another writing on the subject without having personally investigated any of the relatively rare "pure" cases. The conclusion is obvious that the aforementioned investigators looked upon *commotio cerebri* as an incomprehensible and mysterious entity.

In 1853, Fano began his first analysis of the published cases of concussion of the brain and without hesitation he rejected all of these in toto. Then followed an account of a number of personal animal experiments. From these he drew the conclusion that the trauma had given rise to a bulbar hemorrhage. In discussing this subject, Fano repeatedly speaks of the respiratory center situated in the floor of the fourth ventricle. It is surprising to find that Fano neglected entirely to examine his experimental animals for pathological lesions within the bulbus. He merely scrutinized the outside of the bulbus and since hemorrhages generally occurred in this place, he declared the whole idea of concussion of the brain to be "une pure création de l' esprit."

A special commission, appointed by the Société de Chirurgie in Paris, subjected Fano's work to a critical examination. During the discussion, Chassaignac pointed

out that the hemorrhage around the bulbus in Fano's experimental animals was present only when the cranium was fractured. It was decided, therefore, that only the simplest cases of head injuries should be selected for the study of concussion, and to exclude those presenting complications. In this it was hoped to find a group of cases characterized by small diffuse hemorrhages in the brain. Once more one is confronted with the so-called "capillary apoplexy" mentioned by Rokitansky in the first edition (1844) of his textbook. Chassaignac described likewise a case similar to that described by Blandin in 1842.

A characteristic feature of the study of concussion is Fano's neglect of examining the interior of the bulbus, inasmuch as the respiratory center had already been described by Lorry (1760), who severed the spinal cord in the upper part of the cervical region and found that the animal died instantaneously from respiratory paralysis. The exact localization of the respiratory center was established by Le Gallois (1810–11) and confirmed in 1822 by Pierre Flourens. The second edition of the latter's book appeared in 1842 and it is therefore so much more surprising that Fano failed to examine carefully the floor of the fourth ventricle while he was occupied with the action of the respiratory center.

In 1878 Duret began his renowned experimental study of concussion of the brain. In order to prevent the production of oscillations of the cranium, Duret did not strike the animals on the head with a hammer, as had been done by previous experimenters. Through a small trepanation hole in the skull he injected liquid into the epidural space. In this manner he produced a sudden and markedly increased intracranial pressure without transmitting the oscillatory movements from the cranium to the brain, about which so much had already been written, and to which much importance had been attached as being the actual cause of the disorder. By this happy arrangement he succeeded in producing a condition strikingly resembling concussion of the brain in man. Although Duret claimed to have performed very careful autopsies on his animals, he failed to find any significant pathological lesions. In one subsequent experiment he increased the dosage of fluid from 10 c.c. to 100 c.c. and noticed at once such characteristic pathological changes which, in his opinion, solved the riddle of concussion of the brain. This experience has since gained him lasting fame. Duret observed severe and typical hemorrhagic lesions in the floor of the fourth ventricle and around the aqueduct of Sylvius. The floor of the fourth ventricle was torn in such a manner that it was clear that intraventricular pressure had been so great that the injected liquid had torn the walls from within clear out to the surface. This served as the basis for the theory of "le choc céphalo-rachidien." Duret believed that the blow on the head had caused the skull to bulge inwards and thus to exert pressure on the brain surface, which was directly transmitted to the lateral ventricles. Further, that the liquid was driven with such force and speed into the fourth ventricle that the walls and the floor were damaged.

It was already established that the important centers of respiration, vomiting and the vasomotor system were situated in the floor of the fourth ventricle. Thus the cardinal symptoms of concussion of the brain, namely, shallow breathing, feeble pulse and vomiting, could be explained from the lesions found by Duret in the floor of the fourth ventricle in his experimental dogs. Hence the mechanism of concussion of the brain had found an adequate etiological basis.

Immediately Duret's work aroused great attention. In a large work on head injuries published in 1880, von Bergmann devoted a separate chapter to Duret's work, in which he did not embrace completely Duret's theory, because of its inadequate explanation of the loss of consciousness, which is a prominent feature of the clinical picture of concussion of the brain. Duret sought

to explain the loss of consciousness by postulating that the function of the sensory nerves was interrupted by the hemorrhages in the floor and walls of the fourth ventricle. Under these conditions, Duret compared the patient with a chloroform anesthetized person with all impressions eliminated and with only the vegetative centers, especially those controlling respiration and blood pressure, situated in the fourth ventricle, in function. But von Bergmann was fully convinced that the seat of consciousness comprised the entire surface of the cerebral hemispheres. He also believed that an essential parallelism existed between the clinical phenomena and the extent of the anatomical changes which must form the basis of the disorder.

It has been stated already that Duret found only minor lesions in the fourth ventricle, while von Bergmann contended that lesions should be found in the cerebral cortex, which comprised the area of conscious life. However, such changes are rare and when at all present very often slight and occurring in various places. Meanwhile, von Bergmann accepted the explanation of the nature of concussion of the brain advanced by the experimental work of Koch and Filehne and, at this instance, one encounters a grave error in the investigation of this subject. Koch and Filehne, in wishing to avoid the severe lesions produced by striking the animal's head with a hammer, inflicted, instead of a single violent blow, a prolonged series of light taps, the sum of which they believed to be equivalent to one single heavy blow. Believing thus to have produced concussion of the brain in their animals, von Bergmann called this technique "eine glückliche Idée" by means of which all the brain centers were equally affected. However, this belief displays a remarkable lack of critical judgment. In resorting to animal experimentation, in order to shed light on questions in human pathology, one must need reproduce the conditions in man as closely as possible. And frankly speaking, there is no similarity between the *modus operandi* in the experiments of Koch and Filehne and the way in which the human concussion of the brain is produced. Consequently, no anatomic changes were observed in their experimental animals.

The theory of Duret was very shortly mentioned in the textbooks on surgery by König and medical jurisprudence by Hoffmann, respectively published in 1893 and 1903. It is remarkable, nevertheless, that Duret's important theory should have since remained practically unmentioned in medical literature or textbooks of pathological anatomy except in the textbook by Schmaus (6th edition 1901), and in the 11th edition revised by Herxheimer (1915) and N. P. Hansen (1922). Various other authors confine their remarks to the "pure concussion" cases and the assumption of a functional disturbance, or, again, to "capillary apoplexies," etc. and thus leave the subject unsolved.

In his book on concussion of the brain, Theodor Kocher (1901) forms a notable exception, in that he mentions Duret's theory and calls the hemorrhages in the floor of the fourth ventricle, following head injuries, by the name "Duret's lesions," a designation continued by the author in his investigations. Theodor Kocher adopted a reserved attitude towards Duret's theory. He even wished to abolish the term "commotio cerebri" and to replace it by the name "Hirnpressung" (compression of the brain), since he held that the brain was not set in vibration, but that the blow on the head brought about a sudden motion in the brain whereby it became forcibly compressed. Thus he writes: "Der Name Hirnpressung würde ganz entsprechen und die falschen Vorstellungen ausmerzen, welche mit dem alten Namen Hirnerschütterung verwachsen sind." (The name "brain compression" would be quite appropriate and would root out the false conceptions that have grown up around the old name of "concussion of the brain.") Since that time both Duret's name and theory have fallen into oblivion in the German litera-

ture. Thus the renowned pathologist Bollinger at Munich, the founder of the doctrine of the traumatic "Spätapoplexie," gives an account of Duret's theory of *commotio cerebri*, in his contribution to the international *Festschrift* in honor of Rudolf Virchow (1891). However, he agrees with the medicolegal expert Blumenstock when he says that Duret's theory must remain merely a theory, so long as it is not confirmed by autopsy findings. Up to that time, the only human material to shed light on Duret's theory consisted of 3 or 4 cases for which Duret was indebted to his French colleagues and which are rather cursorily described in his doctor's thesis in 1878. The appeal of Bollinger and Blumenstock to investigate *commotio cerebri* was not complied with until the author published his first work in 1930 on the traumatic brain injuries. This study was followed with a larger work in 1933, issued among the publications of the Norwegian Academy of Science, and which is apparently the first systematic investigation of this subject. In the literature one encounters nothing but scattered case reports. Even the French literature fails to reveal any investigation on persons dead from head injuries in which a search has been made for the pathological lesions described in Duret's experimental studies. This fact is clearly established in Forgue's textbook on surgery (1928), which only refers to the few human cases studied by Duret in 1878.

The author's interest in this problem arose from a dispute about an accident insurance case in which he was acting in the capacity of expert medical advisor to the counsel for the relatives of the deceased. This law suit made the author clearly understand how extremely divergent opinions might be regarding the significance of postmortem findings in such a case. The eight professors called as medical experts by the insurance companies unanimously agreed that the hemorrhage in the fourth ventricle had arisen spontaneously. The author referred to the literature from around 1880 and contended that the head injury could have given rise to the hemorrhage in question and the death of the deceased. From the moment of this dispute the author began systematically to collect material bearing on the subject and shortly he came to the conclusion that "Duret's hemorrhages" occurred frequently in the floor of the fourth ventricle in persons dead from head injuries. When the aforementioned lawsuit was settled and the criticism occasioned by the author's first publication had subsided, he continued investigations in this little explored field, because of the practical and scientific importance inherent in Duret's theory. Last autumn he was able to submit an account of 42 cases of concussion of the brain personally investigated. The macroscopic examination alone revealed more than 60 per cent of hemorrhages in the floor of the fourth ventricle and the microscopic examination clearly demonstrated that practically all persons presented such hemorrhage. This startling finding made the author commit himself to state that practically all persons who die as the result of head injuries show more or less extensive hemorrhages in the floor of fourth ventricle, in the immediate locality of the vital nerve centers of respiration and the vasomotor system.

These pathological results suggest the following relevant questions, which call for a brief discussion: firstly, whether the observed hemorrhages caused the death, or, secondly, if Duret's theory is correct as regards the etiology of the hemorrhages. The answer to the first question may be that the hemorrhages are rarely extensive enough fatally to damage the nerve tissue. Since the hemorrhages are mostly of moderate dimensions, it is perhaps more correct to say that the hemorrhages are the morphological expression of the circulatory disturbances produced in this part of the central nervous system. The proper functioning of the central nervous system necessarily demands unimpaired circulation and hence death follows inevitably as

a direct consequence of impaired blood supply. As far as the second question is concerned, one must pay attention to the constructive side of the theory of Duret, which the author feels to be in some degree too rigid. He believes, however, that the theory contains a grain of truth, although much work is still required to elucidate the mechanism of the hemorrhages.

In dealing with persons who have sustained severe head injuries, one should always consider the possibility of hemorrhages in the bulbus, around the vital centers. This part of the brain is undoubtedly a *locus minoris resistentiae* in case of head injuries, irrespective of whether the hemorrhages arise in the way Duret thought or in some other manner. The function of the anatomist is not to advance any opinion on clinical questions. The fact remains, nevertheless, that these findings suggest caution as regards operations on patients with severe head injuries.

The author's results support the theory of Duret concerning the etiology of *commotio cerebri*, inasmuch as an agreement exists between Duret's experimental research and the author's postmortem findings in persons dead from head injuries. As an immediate consequence of cranial injury, the patient presents as a general rule a characteristic picture of concussion of the brain, which if he survives for a few days gradually subsides. It seems natural, therefore, for the author to assume that a relationship exists between the nearly constant presence in such cases of hemorrhages in the floor of the fourth ventricle and the clinical symptoms and signs, and that the same importance should be attributed to the postmortem findings as to the results of the experimental research. This seems so much more justifiable in view of the fact that the ventricular hemorrhages are always present in persons who die within a few minutes after the accident occurred.

The criticisms levelled against the author's studies concern the "one-sidedness" of the material examined and the author's right to draw broad conclusions from it. The material studied consists of that usually offered by a large city hospital dealing with all sorts of accident cases, among which one encounters many automobile accidents. Some of these fatal head injuries were produced by automobiles not always running with a speed of 30 to 50 miles an hour and hence the violence should be considered moderate. In other instances of accidents the violence inflicted was very slight in some cases and extremely severe in others. In brief, the material examined consists of a cross-section of the usual head injuries.

Another objection has been advanced that medicolegal pathological material is better suited to throw light on questions that arise in connection with head injuries. Thus, there have been submitted data comprising 26 medicolegal autopsies in cases of head injuries which showed a complete absence of hemorrhage in the floor of the fourth ventricle. The material in question was collected during approximately the same period in which the author's 42 cases were studied in which he encountered nearly 100 per cent hemorrhages in the floor of the fourth ventricle. A greater discrepancy between two parallel series of investigations scarcely exists. The question arises whether or not this disagreement is due to the fact that the violence causing death in the medicolegal cases was slighter than in the author's material, since no hemorrhages occurred in the former.

In carefully examining the detailed description of the manner in which death was produced in the 26 medicolegal cases it is difficult to accept such an explanation. It is stated for instance that one victim was killed following several severe axe blows on the head. Another engaged in a fight and was struck on the head with a wooden stick. A third was struck with the fist and afterwards with an iron automobile crank. A fourth was thrown off a motor truck. A fifth got his head crushed by a motor bus, etc. It is difficult to pretend that the head injuries sustained

in the medicolegal cases should not have occasioned the same pathological lesions as those observed with constancy in the author's series. One is compelled, therefore, to seek a different explanation of these striking divergences. Such is most likely found in the manner of performing the autopsy. The author has made it a rule never to section a brain which has not been hardened by fixation. He finds the fresh brain too soft and unmanageable for a satisfactory examination. In most pathological laboratories it is customary to section the brain immediately after its removal from the skull. In this manner many important details are overlooked. Even with the best precaution and clean knives, fresh blood will be dragged down across the cut surfaces. The advantage of hardening the brain before attempting to section it is obvious. With this practice, it is needless to state that the high frequency of hemorrhages in the floor of the fourth ventricle would long since have been detected in cases of concussion of the brain, and long ago to have confirmed the true etiology of *commotio cerebri*.

Thanks to the work of Dittmar, the position of the vasoconstrictor center in the floor of the fourth ventricle is well known. This corresponds more or less to the site of the facialis nucleus, the area in which the author most often has found extensive hemorrhages. Destruction of this part occasions an instantaneous and marked fall in blood pressure.

One is likewise fairly well aware of the extent of the region in which the respiratory center lies. One knows, at any rate, that this area is larger than Le Gallois supposed. The English physiologist Lumsden demonstrated in 1925 the existence of four co-ordinate respiratory centers in the floor of the fourth ventricle, and that the sectioning of the medulla oblongata at points corresponding to each of the four segments described in his scheme brings about different types of abnormal respiration. Thus a severance at the upper part of the pons brings about prolonged respiration, while severance in front of, or just at the striae acusticae leads to prolonged inspiratory spasms or cramps in the diaphragm, followed by what he calls "gasping respiration." This place is named the "apneustic" center and the tonic contraction of the inspiratory muscles he terms "apneusis." He also found that the severance at a point farthest back of the striae acusticae occasions instantaneous cessation of respiration. In other words, his experience has verified the existence of the spot which Le Gallois found in 1810 and which Flourens afterwards named "*le noeud vital*." The place of predilection for the hemorrhages seen in persons dead from head injuries is to be found precisely in that part of the fovea rhomboidalis where the circulatory and respiratory centers are situated. It is quite natural, therefore, to meet with clinical signs of disturbance of functions of these centers.

By anology it is also reasonable to believe that the same areas are affected in the "slight" cases of concussion of the brain in persons who soon recover. Either the injury to these patients is so small that no hemorrhage occurs, or else the bleeding is so slight that it is shortly absorbed. The clinical and pathological picture of the "severe" case aids in forming an idea of the state of affairs in the brain of patients who recover from the injury.

In regard to the loss of consciousness in cases of concussion of the brain it is well to recall that opinion has changed about the seat of consciousness from the cerebral cortex to medulla oblongata. In 1914 Bresslauer showed that the surface of the brain is not at all sensitive to blows, while the bulbus is extremely sensitive even to a slight touch. In 1921 Demmer stated that his clinical experience indicated that the traumatic action on the medulla oblongata and on the floor of the fourth ventricle gave rise to the phenomenon of concussion, inasmuch as the center of consciousness was immediately adjacent to this area. Franz Schück, who collaborated with Bresslauer, wrote in 1924 that the trauma leading to loss of consciousness passed through the medulla oblongata, even if the blow was so slight that it was unable to bring about lesions in the cerebral cortex.

"Wird also die Oblongata überhaupt von der Druckwirkung erreicht, so ist eine begleit ende Bewusslosigkeit auf ihre Rechnung zu setzen, ganz gleichgültig, was inzwischen mit der Grosshirnrinde geschieht." (Thus, if the medulla oblongata is affected at all by the pressure, then an accompanying unconsciousness is to be attributed to it, quite irrespective of what takes place in the meantime in the cerebral cortex.) Otherwise Schück adheres to Hitzig's old conception that the whole of the cerebral surface is the seat of conscious functions. "Die Grosshirnrinde in ihrer Gesamtheit gleichgültig ob Sitz der bekannten Funktionen oder 'stumm,' ist Trägerin des Bewusstseins." (The cerebral cortex, whether it is the seat of the conscious or "dumb" functions, is the seat of consciousness.) No individual spot on the surface of the brain stands in specific relation to consciousness and a local disturbance on the surface never occasions a comatose state of insensibility. "Es handelt sich bei dem Problem des klinischen Bewusstseins um das Zusammenwirken bzw. um die gegenseitige Beeinflüssung von Hirnstamm auf der einen Seite, von gesammt er Grosshirnrinde auf der anderen Seite." (Concerning the problem of clinical consciousness it is a question of the collaboration or reciprocal influence of the brain-stem and the entire cerebral cortex. If the whole of the cerebral cortex is put out of function, while the subcortical centers in the brain-stem are preserved, the most profound coma will not occur, but only that condition known from experiments on the decerebrate animals. "Zum Bilde der tiefsten klinischen Bewusstlosigkeit ist stets auch ein Betroffensein der subkortikalen Zentren bzw. des Hirnstamms erforderlich." (To produce the picture of the deepest clinical unconsciousness the subcortical centers or the brain-stem must also be affected.)

Bresslauer's experiments have, it is true, been criticized by Knauer and Enderlein in 1923, but Schück in 1924 maintained the correctness of Bresslauer's and his own views. He repeated in 1928 that the deep unconsciousness in cases of head injuries is connected with the conditions in the medulla oblongata, and he concluded by saying that he believed that the medulla under such circumstances is subjected to an "acute compression." In the same work he maintained his belief in the traumatic brain-stem theory. But like Fano, he had not the slightest idea about the hemorrhages constantly present in the bulbus under these circumstances. For the present, the author himself does not want to draw any conclusions about the significance of his pathological findings in their relation to the etiology of the state of unconsciousness. He is desirous to revert to the "pure" cases, however, which formed the starting-point for this discuscussion and which are rarely seen. The question arises about Duret's attitude towards such "pure" cases. It is quite clear that the founder of the new theory of the etiology of concussion of the brain was not inclined to reject the possibility of "pure" cases. Even as it is accepted that a person may die from a sudden fright, for instance as the result of a "psychic trauma," one may equally well imagine that a mechanical factor may possibly lead to the same fatal disturbance of function as was caused by fright. As far as Duret's opinion is concerned, he believed the actual occurrence of such a case to be infinitely small. Before he would accept the reports of such a case he demanded a careful examination of the entire bulbus. Thus it may be said that Duret died in 1922 with a challenge to pathologists to examine the medulla oblongata in cases of concussion of the brain, to settle the question whether the so-called "pure concussion" case did not present pathological lesions.

CONCLUSION

The histopathological material of several cases of concussion of the brain suggests that an etiological relationship exists between the clinical symptoms and signs and fatal issue of this disease and the constant presence of more or less extensive hemorrhages in the floor and walls of the fourth ventricle, in the immediate vicinity of the vital centers of respiration and the vasomotor system.

BULLET EMBOLISM*

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IN time of war, the entrance of bullets or fragments of projectiles into some part of the vascular system with impaction of these foreign bodies in a more or less distant, narrower part of the system is not uncommon. This was the experience of Borst during the World War, according to Henes.[1] In civil life this occurrence is naturally a rarity. Nevertheless, enough cases are on record in the literature, reported on the basis of peace time experience, to warrant a consideration of the possibility of this type of embolism by those diagnosing or examining at autopsy cases of shooting. Thus both surgeon and medical examiner will be interested in the following case report.

When a bullet enters the body, it may follow a devious route because of its impingement on organs or tissues of varying density and at varying angles. Hence a study of the wound of entrance and the immediately subjacent track may be of little aid in locating the resting place of a bullet. This becomes more complicated when the bullet enters the vascular system and passes to some other part of the body. Thus Rubesch[2] stated with regard to injuries by projectiles that pain in an extremity well removed from the primary injury might well suggest the possibility of embolism caused by the projectile. Similarly, in searching by roentgen ray or at postmortem for a bullet which has no wound of exit, attention should be given to the arteries of the extremities if the bullet is not located in the trunk region.

REPORT OF CASE

A Negro, aged twenty-three, received a pistol shot in the right flank. Approximately an hour later he had medical attention. The blood pressure showed a systolic reading of 60 mm. Hg. The diastolic pressure could not be obtained. The pulse was rapid, about 140 beats per minute, and weak. The mucous membranes were pale. The skin was cold and clammy. The right leg was limp and the patient complained of pain in it. There seemed to be no sensation in either leg.

The bullet wound was located in the posterior axillary line between the tenth and eleventh ribs.

The pulse remained weak and could not be counted. The blood pressure dropped. The patient died slightly more than four hours after being shot.

The autopsy, performed at Duke Hospital, showed a sturdy young Negro. The wound of entrance of the bullet was in the right flank near the tip of the eleventh rib. The hole was 1 cm. in diameter. No wound of exit could be located. The abdominal cavity contained approximately 150 c.c. of blood. A large retroperitoneal hemorrhage surrounded the right kidney and extended medially tothe aorta.

Dissection near the right kidney allowed the path of the bullet to be traced (Fig. 1). The fragments of the tip of the eleventh rib lay at the point of entrance of the bullet. The lower pole of the right kidney showed a groove, 1 cm. deep, where the bullet had passed. The right ureter had been severed. The right iliopsoas muscle showed a groove which led to a wound on the right side of the body of a lumbar vertebra. When the aorta was opened, a horizontal slit was noted extending through the entire wall of the aorta and two-thirds of the distance around this vessel, sparing only part of the right side. The tear lay 3 cm. above the bifurcation of the aorta and included the orifice of one of the right lumbar arteries.

When the right common iliac artery was laid open, the scissors struck the bullet firmly lodged in the orifice of the right external iliac artery. The bullet measured 9 mm. in width

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and 16 mm. in length and had a longitudinal groove on one side. When the greatly contracted segment of external iliac artery below the point of embolism was opened from below upward the flat rear end of the bullet was encountered. The right internal iliac artery was patent.

The inferior vena cava was uninjured. Dissection showed that the large nerves to the right leg were all intact. The wound in the vertebral body was superficial and the cauda equina appeared normal. The muscles and other tissues of the right leg did not differ in gross appearance from those of the left leg.

In addition to the changes caused by the bullet, the macroscopic and microscopic study of the case showed the following incidental abnormalities: calcified mediastinal and peribronchial lymph nodes; disseminated tubercles in liver and spleen; healed ulcer of glans penis; healed inguinal lymphadenitis; focal atrophy of the testes.

COMMENT

The complete course of the bullet was evident. The missile had passed through the retroperitoneal tissues to a vertebral body. Here it had been deflected anteriorly and had been caused to enter the abdominal aorta. Its force largely spent, the bullet was seized by the column of moving blood, and also probably acted on by gravity, and carried as an embolus to the bifurcation of the right common iliac artery where it lodged, not as a "riding" embolus at the bifurcation, but in the mouth of the right external iliac artery. The bullet must have been turned around somewhere in its course, because the nose pointed upstream.

The symptom of limpness of the right leg must have been due to the ischemia produced by blockage of the iliac artery, since no nerve injury occurred, and since the other leg was not limp. The pain in the right leg may be similarly explained.

The clinical observation that there was no sensation in either leg is harder to explain. The cauda equina was intact, but there might have been injury to the posterior root ganglia at the intervertebral foramina due to the force with which the bullet struck the vertebral body.

The chief cause of death was apparently the loss of blood into the retroperitoneal tissues from the torn aorta, the injured lumbar artery, and the traumatized right kidney.

Fig. 1. Embolism of an artery by a bullet. Bullet, deflected forward by a vertebral body, entered aorta and passed downward to become wedged in right external iliac artery.

Mention of embolism by bullets is rare in American medical literature. Recently, however, Gonzales[3] wrote: "Not infrequently a bullet will enter a blood vessel and be carried along in the blood stream. This occurred in one case, in which the missile entered the thoracic aorta and was found in the left iliac artery."

Most of the articles relating to embolism by projectiles are in the German language. A good account of this kind of embolism was given by Rubesch[2] in 1912. He collected 17 cases, including one of his own. In some instances it appeared that the missile had become "embolic" after death, as a result of moving the body. He divided his cases into the following five groups:

1. Transport of the projectile against the current of blood, probably a postmortem occurrence, 3 cases.

2. Transport with the blood stream, possibly a postmortem occurrence, 5 cases.

3. Genuine embolic passage, 7 cases.

4. Passage during life, in part against the current of blood and in part with it, 1 case.

5. Secondary intravital passage, that is, the dislodgment of the projectile from its first resting place, and transport by the blood stream during life.

Of the intravital cases the projectile entered through the heart in 4, through the pulmonary veins in 4, through the aorta in 2, through the crural artery in one, through the subclavian vein in one, and through the pulmonary artery in one. The longest pathway traversed in the blood stream was from the pulmonary vein to the femoral artery.

In my case "embolism" after death can be excluded because of the pain and limpness of the right leg not long after the shooting occurred.

Paltauf,[4] reporting in 1933 a case of embolism of a branch of the pulmonary artery by a bullet, found 31 cases of bullet embolism in the literature, of which 4 involved the pulmonary artery.

Since in my case the bullet came to rest in the right external iliac artery, it is interesting to note the conclusion of Walcher[5] that the left iliac artery is favored by a bullet entering the aorta. In the literature he found 6 instances in which the projectile had gone to the left at the aortic bifurcation and only one instance in which it had gone to the right. The explanation given was that the left common iliac artery bends less sharply away from the longitudinal axis of the aorta than the right one does.

The embolic projection of metal foreign bodies is interesting with regard to the physical relationships involved. The specific gravity of a bullet is far beyond that of an embolus composed of thrombus material. Hence the bullet would be less easily moved by the blood current. The velocity of a column of blood would have to be indeed great to support a lead bullet. However, a bullet entering the aorta of an individual who was upright would be acted upon by gravity and carried downward. If a bullet entered the heart of an individual who fell to a horizontal position, the blood stream would need merely to roll the bullet along the vessel.

Embolism with metal foreign bodies has been produced experimentally in animals. Schloffer[6] placed shot 2 mm. in diameter in the inferior vena cava of each of two rabbits. One died within an hour. The other was killed after about the same interval of time. The shot were located in the larger branches of the pulmonary arteries. No mention is made of the position of the animals during the experiment. Riethus[7] placed two shot, chained together, into the jugular vein of a rabbit and seventy-seven days later found the shot caught in the trabeculae of the right ventricle of the heart.

An article by Henes[1] in 1919 indicated that, according to the reported cases, the correct diagnosis of projectile embolism had been made only three times. In two of these the foreign body had been successfully removed. In the third case, that of Rubesch,[2] gangrene of the extremity ensued because extensive secondary thromboses formed before removal of the bullet.

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CASE REPORTS

RECOGNITION OF RADIOPARENT FOREIGN BODIES AT THE PERIPHERY OF THE LUNG*

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THE purpose of this communication is to briefly review a caxe of foreign body lodged at the periphery of the lung and to call attention to an association of symptoms and signs which might have a definite diagnostic value in similar cases. The case in question presented a difficult diagnostic problem. If the correct solution had been arrived at earlier, a definitive program could have been instituted from the start, with every prospect of ultimate success.

It is unnecessary to review the clinical and roentgenological signs on which a diagnosis of foreign body in the airways is ordinarily based. It is also unnecessary to review the roentgenological findings in cases in which a radiopaque foreign body has reached the periphery of the lung. It should be emphasized, however, that radioparent foreign bodies are recognized, roentgenologically, by the secondarily induced pulmonary pathology. The principal pathological changes are obstructive emphysema, atelectasis, "drowned lung," pneumonitis and abscess. All of the foregoing conditions have definite roentgenological characteristics. But to produce such changes, the foreign body must occlude, in whole or in part, one of the larger bronchi.

Some foreign bodies are of such size and nature that they may migrate rapidly to a peripheral position. If they are radioparent, they cannot be directly visualized and the secondarily induced pathology will no longer be lobar in its distribution, but lobular only, or limited to a sector of the lobe. In the following case all pathology was caused by a peripherally placed radioparent foreign body.

CASE HISTORY

The patient, H. B. Jr., was a thirteen-year-old schoolboy. When admitted to the Arnot-Ogden Memorial Hospital on November 9, 1933, he had been sick for three weeks. Early in this sickness he had coughed up frank blood and recently there had been a series of increasingly severe pulmonary hemorrhages. His past health had been good. There was no history of trauma. There was no history of a foreign body having been aspirated. Physical examination showed a well developed, pale, sick-looking boy. Except for the findings in the right lower chest, the examination was negative. In the right lower chest, posteriorly and laterally, there were dulness, harsh breath sounds, slightly prolonged expiration and moist râles. The upper respiratory tract was negative. Roentgen examination showed an area of slightly increased density extending fanwise from the right hilum to the costophrenic angle. This area did not fill with iodized oil, 40 per cent, after supraglottic introduction. The lateral plate showed this unfilled area to lie posteriorly and to measure approximately 8 cm. in diameter. There was no evidence of an

* From the Chest Clinic, Arnot-Ogden Memorial Hospital, Elmira, N. Y. Read before the New York Society for Thoracic Surgery.

opaque foreign body and no suggestion of a non-opaque foreign body. Bronchoscopic examination was negative except for the airways of the right lower lobe. They contained blood-tinged secretions and their mucosa was slightly inflamed. No foreign body was visible. Examination of the sputum and bronchoscopically obtained secretions was negative for tuberculosis bacilli, but did show many streptococci and staphylococci, as well as a few spirochetal organisms. There was no evidence of any blood dyscrasia.

During the period of initial examination another severe pulmonary hemorrhage occurred. Without further delay a right therapeutic pneumothorax was instituted and the bleeding effectively controlled. For four months the therapeutic pneumothorax was continued. During this time the boy was at home and remained apparently well; his weight increased from 110 pounds to 126 pounds. There was no cough; there was no recurrence of hemoptysis; but fluoroscopically the same fan-shaped shadow remained visible in the right lower lobe.

Suddenly, in March, 1934, the pulmonary hemorrhages recurred. The patient was readmitted to the hospital. Within four days there were ten severe hemorrhages. Pneumothorax no longer controlled them. Bronchoscopy confirmed the source of bleeding to be in the right lower lobe. Two transfusions of 500 c.c. each were given on March 21 and 22. On March 23, 1934, a one stage lobectomy was performed according to the Brunn-Shenstone technique, and this was immediately followed by a third transfusion of 500 c.c. The patient died the same day.

Examination of the excised lobe showed a small abscess cavity situated in the extreme periphery and in this cavity lay a foreign body 1.75 cm. long, which appeared to be rotted wood. The surrounding lung tissue was infiltrated with blood, and no longer compressible or collapsible.

FINAL COMMENT

In retrospect, it is easy to realize that an exploratory thoracotomy would have disclosed the inflammatory area in the right lower lobe. It would have been easy to have delivered this portion of lung outside the chest wall and to have secondarily drained it. The foreign body would have been discovered and with its removal, complete recovery would have promptly ensued. It was no easy thing, however, in the absence of a definite diagnosis, to advise a surgical program instead of the simple expedient of therapeutic pneumothorax. What might have helped toward an accurate diagnosis?

Three symptoms and signs deserve consideration. Hemorrhage was the dominant symptom from the onset. Low grade suppuration was also present. Of still greater significance was the fact that the area of inflammation was limited to a small sector, was constant in its localization and was not migratory. The suggestion, therefore, is offered that the association of hemorrhage and of a low grade, constantly localized inflammatory reaction, lobular in its distribution, should demand the consideration of peripherally placed foreign body in arriving at a differential diagnosis. If all other possibilities can be excluded, then association of hemorrhage and constant non-migratory lobular inflammation should be a rational basis for a search for such a foreign body, either by endoscopic or transthoracic exploration.

OSTEOMYELITIS OF THE PATELLA*

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WHILE osteomyelitis of the patella is a rare condition, it must be considered as a possibility in the differential diagnosis of infections about the knee-joint.

The earliest case report was that of M. Thirion in 1829. Ducuing reviewed the French literature in 1911. In 1917, Walther gave an account of 10 cases from the literature and 2 of his own from Lexer's Clinic, apparently unaware of Chalaby's report of 22 cases in 1914. While Cheskey reviewed 35 cases, including his own, in 1922, the more complete review was that of Rocher in the same year, reporting 50 cases from the literature and his own case. Of this series, apparently only 5 cases were chronic, the remainder acute, and in the main, involving the patella primarily. Both of these authors failed to include Rosenbach's case of acute osteomyelitis of the patella which he reported in 1922. Since 1923, Mariupolsky has reported 2 cases of chronic abcess (Brodie) of the patella (1930), while Manclark (1933), Christopher (1933), and Sagel (1934), have each recorded a case of acute primary patellar osteomyelitis.

Patellar osteitis may be due to direct implantation of infection, but is most commonly of hematogenous origin, at times secondary to some far-removed focus or intercurrent infection, and frequently associated with slight trauma (50 per cent of cases in Rocher's series). The infecting organism is usually the staphylococcus or streptococcus.

No cases are recorded before the age of five, few after fifteen, the majority occurring between these age limits, i.e. during the period of greatest vascularization of the patella. The infection may lie dormant for many years, to be lighted up by some intercurrent condition or during lowered resistance.

There is pain, at first mild, later severe and lancinating, and referred to the knee, moderate fever and leucocytosis, rapid pulse, and swelling of the anterior and lateral aspects of the knee with maximum tenderness localized over the patella. There is often an effusion of the knee-joint. The leg is held fully extended until the joint effusion leads to partial flexion.

The clinical picture closely simulates that of patellar bursitis, suppurative and non-suppurative arthritis of the knee-joint, and osteomyelitis of the lower end of the femur. The roentgenogram is of great assistance, although the diagnosis is frequently in doubt until a sequestrum forms. It must be differentiated from primary tuberculosis of the patella and patellar osteochondritis.

The treatment consists, early, of incision and drainage, and the removal of sequestra, care being taken to preserve the periosteum and posterior cartilage; posterior splintage and physiotherapy. If the joint is involved, arthrotomy is essential, and if ankylosis is inevitable, the proper position of the leg must be sought for.

The following case report brings the total number of reported cases to 58:

H. T., a female, aged twenty-eight, was admitted on August 29, 1934, complaining of

* From the orthopedic service of Dr. J. Torrance Rugh, Jefferson Hospital Philadelphia.

pain and swelling in the left knee, of acute onset and of eight weeks' duration. There was no history of any trauma, but the patient recalled a slight, suppurating infection of the left hand due to an insect bite at about the time of the onset of her present illness. The condition had been treated by a physician as an acute arthritis. Two weeks prior to admission, through a small incision over the patella, some seropurulent material had been obtained, and a draining sinus had resulted, persisting at the time of admission. An *x*-ray study at that time had apparently been negative.

Fig. 1. Right knee on admission, October 29, 1934.

Fig. 2. Left knee on admission.

Examination of the left knee revealed swelling, redness and tenderness, most marked over the patella, with a draining sinus on the antero-lateral aspect. The knee was held in slight flexion, with motion limited. There was an effusion into the knee-joint, but no evidence of ankylosis. The patella felt roughened on its anterior surface.

The temperature on admission was normal, rose to 100° several days later, to return and remain normal thereafter. The leucocyte count was 12,000 with a polynucleocytosis. Blood serological tests were negative.

The roentgenographic study shortly after admission revealed localized changes in the left patella indicative of an osteomyelitic process (Fig. 2). The right patella was negative (Fig. 1).

A conservative regime of treatment was pursued. Because of her moderately septic condition and a secondary anemia the patient received three small transfusions of whole blood by the direct method.

On September 19, the roentgenogram revealed increasing destruction of the left patella, with diseased tissue closer to the skin surface (Fig. 3).

On October 2, a small sequestrum ($\frac{1}{2} \times \frac{1}{4} \times \frac{1}{4}$ inches) was extruded through the sinus. Another roentgenogram (October 8) revealed a subsidence in the inflammatory process of the left patella, with evidence of an area on the anterior surface from which the sequestrum had apparently detached itself (Fig. 4).

A few days later, one of the authors (J. R. M.), under nitrous oxide-oxygen anesthesia, curetted the patella through a small incision on its lateral aspect, removing several pieces of diseased bone and much necrotic debris (culture of which revealed a growth of

staphylococcus albus), packing the wound with vaselinized gauze.

The wound at the time of the patient's discharge on November 6, 1934, was filling with abundant healthy granulation tissue. The disappearance of all symptoms and signs, with normal temperature and leucocyte count, indicated a complete subsidence of the pathologic process. Follow-up *x*-ray studies have evidenced satisfactory subsidence of the pathological process.

Fig. 3. September 19, 1934.

Fig. 4. October 8, 1934, six days following spontaneous extrusion of sequestrum.

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PERCUSSION TREATMENT FOR FRACTURES OF THE LEG WITH DELAYED UNION*

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THREE cases are presented in which the percussion treatment for delayed union or non-union of fractures of the leg was successfully used after the usual forms of palliative treatment had failed. In each instance the roentgenograms showed a persistence of the fracture line with sclerosis of the bone ends. The patients had pain and clinically, there was little or moderate motion at the site of fracture.

The patient is confined to bed and the fractured part is supported in a plaster shell. Once each day, the plaster shell is removed, the patient is placed on a table with the foot over the edge and the foot and ankle are supported so that the fractured fragments are in proper alignment. The sole of the heel is tapped or percussed about a hundred times with the ulnar border of the hand and with a wrist motion.

This procedure constituted the sole treatment in the 3 cases herein reported and union usually resulted in about six weeks.

CASE 1. J. B., a structural steel worker, thirty-five years of age, on January 13, 1930, had sustained a compound comminuted fracture of both bones in the lower third of the right leg when three iron beams 18 inches across and 15 feet long rolled onto his leg. Two closed reductions and one open reduction were attempted without success. I first saw the patient four months after his injury on May 12, 1930. His general health was good. His Wassermann and other blood studies were negative. He walked with the aid of two crutches and bore no weight on the injured extremity. Clinical examination showed a compound infected fracture of both bones of the right leg in the lower third. A small amount of very foul-smelling seropurulent discharge was present. Two loose fragments of bone, denuded of periosteum, capped the upper end of both lower fragments. *X*-ray examination showed a fracture below the mid third of both bones of the leg with non-union. Several sequestra were present.

On May 26, 1930 a sequestrectomy was performed. The wound was packed and a plaster applied, after the method of Orr. The plaster was changed about every six weeks until February 12, 1931, when the patient was fitted with a walking caliper brace.

Examination on May 15, 1931, sixteen months after the injury, showed that non-union was still present. *X*-rays showed the fracture line clearly. The lower end of the upper tibial fragment was sclerotic. Clinically, there was motion at the site of fracture and weight bearing, in the brace, caused discomfort at the site of fracture. The percussion treatment was started at this time. The sole of the heel was tapped three hundred times once a day. The leg was supported in a plaster shell between treatments and the patient was kept in bed.

On July 14, 1931, two months after the percussion treatment started clinically there was solid union of the fracture. *X*-ray examination showed an exuberant callus about the fragments and a considerable permanent callus between the fragments. The crutches were discarded but the caliper brace was continued. The last *x*-ray taken was made ten weeks later, on September 28, 1931 or four and a half months after the percussion treatment began. It showed that the fracture line was not entirely eradicated but the callus was denser and had increased in amount. Three months

* Dr. Frank Ober, of Boston, who suggested the treatment, was advised of it by his associate, Dr. Brewster, who learned of it from the late Dr. William Baer, of the Johns Hopkins Hospital.

later on examination the patient stated he could be on his feet eight to ten hours a day with comfort. He was able to walk up steps on his toes. He had no discomfort at the fracture site and the brace had been discarded. The right lower extremity was ¾ inches shorter; the foot was in 115° of equinus with a moderate restriction of lateral motion. The knee flexed to 90°.

The patient was re-examined in July of 1934. The union was solid. He had returned to his former occupation as a structural steel worker. Motions of the foot and knee were essentially normal. He wore a ¼ inch tap and heel lift on the right shoe.

Case II. F. C., a man, aged sixty-five years, was seen in consultation with Dr. J. A. Perkins of Coatesville and it is with his permission that this case is being reported. At my suggestion he instituted the percussion treatment. The patient was seen on September 25, 1933. A year previously he sustained a compound fracture of the lower third of both bones of the left leg. He walked with the aid of two crutches, bearing some weight on the injured extremity but complained of pain and a sense of insecurity.

Examination showed a thickening of the left leg which was slightly bowed and ⅞ inch shortening as compared with its fellow. Forcible manipulation caused pain at the site of fracture and slight motion was discernible. *X*-ray examination on August 19, 1932, soon after the injury, showed beginning callus formation in both bones, while *x*-ray films made three months later showed no callus formation at the tibial fracture. Callus which had started to form, as shown in the August roentgenograms, had evidently been absorbed. The bone ends were sharply defined. An *x*-ray made in March or six months after the injury showed a sclerosing process at the fractured ends of the tibia[1]

Fig. 1.

Fig. 2.

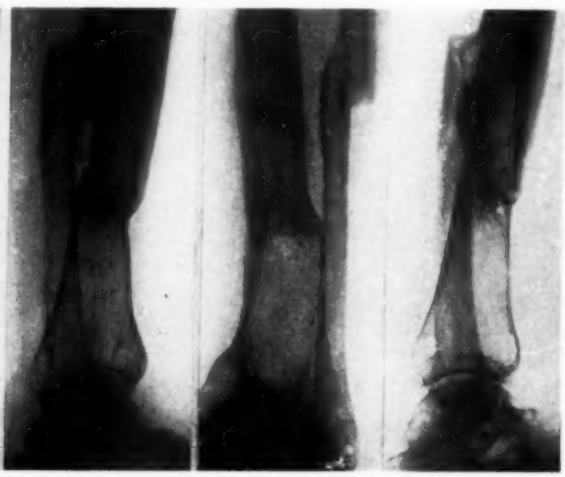

Fig. 3.

fragments and union was not present.

X-rays dated September 25, 1933, a year after the injury (Fig. 1) showed good union of the fibula. The fracture line of the tibia was still visible and a slight sclerosis was seen in the lateral view, at the posterior half of both tibial fragments. Rest in bed, the wearing of a posterior plaster shell and percussion of the sole of the heel one hundred times a day for six weeks was advised.

The patient was seen again four months later on January 16, 1934. The treatment had been performed by Dr. Perkins as above outlined for a six weeks' period. Clinically union was solid. The patient walked with the aid of a cane. He discarded his crutches when the treatment was started. *X*-ray examination (Fig. 2) showed much less sclerosis at the site of fracture. Permanent callus was bridging the tibial fragments. The patient was last seen on October 24, 1934, thirteen months after the percussion treatment started and more than two years after the fracture. His progress had been uneventful. He had ⅞ inches of shortening and wore a corrective shoe. *X*-ray examination on October 24, 1934 (Fig. 3) showed an almost complete obliteration of the fracture line with good bony healing and solid union.

Case III. M. F., a girl aged seventeen, was struck by an auto on March 6, 1933. She had a comminuted fracture of the mid third of both bones of the right leg. The fracture was reduced and plaster applied, which the patient wore for ten weeks. The final note on her

surgical clinic chart read that clinically there was greater firmness at the site of fracture but the *x*-ray shows no callus.

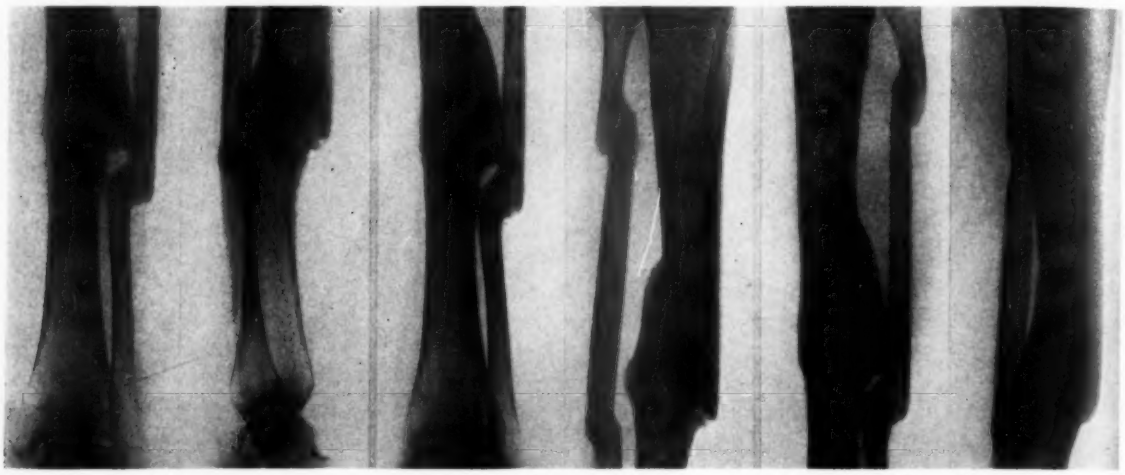

FIG. 4. FIG. 5. FIG. 6.

The patient was referred to the Orthopedic Clinic at the Jewish Hospital on September 8, 1933. The patient walked well without support, the body was listed to the right. She complained of pain at the site of fracture only on activity. On weight bearing the patient was conscious of slight motion at the site of fracture from side to side and from before backward. Clinically there was a slight amount of anteroposterior motion just below the mid third of the right leg. The right lower extremity was 1 inch shorter.

X-ray examination following the injury on March 6, 1933 showed a transverse fracture below the head of the right fibula and another below the mid third with a half inch overlapping. There was a comminuted fracture of the mid third of the tibia with a 50 per cent internal displacement of the proximal fragment.

X-ray examination of the fracture when the patient was referred to the Orthopedic clinic on September 8, 1933 (Fig. 4) showed a fracture of both bones of the right leg. The fibular fragments were overlapping a half inch and were firmly united. The line of fracture in the tibia was still visualized. The proximal fragment was displaced 10 per cent in the anteroposterior view and 25 per cent in the lateral view. A sclerotic process at the site of fracture was present.

The patient was admitted to the Orthopedic Ward at the Jewish Hospital on September 18, 1933 for the percussion treatment with a diagnosis of a fracture of the bones of the mid third of the right leg with a delayed painful union of the tibia. The treatment consisted of tapping the sole of the heel 300 times once a day, rest in bed and in a plaster shell for six weeks. The patient was allowed out of bed and to walk two days before her discharge from the hospital on November 4, 1933. Subjectively and objectively there was no motion at the site of fracture and walking caused no discomfort. The patient was seen six days later and stated that she was quite active and was free from pain and discomfort. *X*-ray examination on November 3, 1934 (Fig. 5) showed possibly some added callus formation. The picture was poor.

The last examination was made June 22, 1934 about six and a half months after her discharge from the hospital. She walked well wearing a high heeled shoe with an inner heel wedge. The pelvis had tilted to accommodate for the shortening. She had no discomfort in the previously injured right lower extremity. All motions of the joints of this extremity were free and unrestricted. There was a shortening of one inch. Clinically and on *x*-ray examination (Fig. 6) union of the fracture was firm and solid.

Mommsen[1] devised an apparatus permitting the patient to release a hammer to strike the sole of the heel through a window cut in a plaster. This was done for about

[1] Mommsen, F. Mechanische biologische Gesichtspunkle für die unblulige Behandlung der verzogerten Fraktenheilung und Pseudoarthrose mit Angabe einer biologischen bihandlunges Methode. *Ztschr. f. Orthop. Chir.*, 51: 472–88, 1929.

ten minutes of every waking hour for one or two months. He reported 4 cases in which healing occurred in fractures of the leg with non-union. Krida[2] used Mommsen's method; a modification of the hammer and dam method of Thomas. Krida reported one case in which healing of the fracture of the leg occurred in two months.

The accompanying explanation is my own thought as to the pathology present and the manner in which union occurred:

A non-union or delayed union occurred. Undoubtedly, because of the delay in healing a fibrosis of the soft tissues about the site of fracture and a sclerosis of the bone ends resulted, even as dense scar tissue forms about a slow-healing osteomyelitic wound. This fibrosis caused a disturbance in the nutrition and blood supply and further retarded healing. The percussion treatment stimulated union by causing a submaximal[3] irritation at the site of fracture. This irritation may lead to local changes at the fracture site favoring new bone formation. This may be due to an increase in the local blood supply, a change in the pH or a proliferation of normal connective tissue. It is the same type of change which results from weight bearing in delayed union except that in the latter case the stimulation is carried to excess. It is stimulation, plus rest, which seems to be effective.

[2] Krida, A. Mommsen's method of submaximal stimulation for tibial pseudoarthrosis. Am. J. Surg., n.s. 10: 366–368 (Nov.) 1930.

[3] Rest in plaster was considered as a minimum stimulation; walking in plaster a maximum stimulation.

CONCLUSION

The percussion treatment for delayed union, or perhaps non-union of fractures of the leg is a palliative treatment worthy of trial before resorting to surgery. It is particularly worthy of a trial in those patients in whom radical surgery is being delayed until all possibility of infection at the site of an old inflammatory lesion or infected area has abated. In the 3 cases herein reported, there was sclerosis of the fragments at the site of fracture, a fibrous union was present, but the patients all had pain and disability. Two of the patients used crutches and one could bear no weight on the injured extremity. In all 3 cases the fractures had been compounded. The ages of the patients were thirty-five, sixty-five and seventeen years. The interval between the time of the fracture and the beginning of the percussion treatment was sixteen months, twelve months and six months respectively. Firm union was obtained in all 3 cases.

SUTURE OF RUPTURED SUPRASPINATUS TENDON

FOUR DAYS AFTER INJURY*

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IN Codman's recent book[1] on the shoulder, he again calls attention to the importance of rupture of the supraspinatus tendon as the cause of many prolonged cases of shoulder pain and disability. He emphasizes the advantage which might be expected to follow the early recognition of the condition and suture of the tendon. However, in his twenty-five years' experience and in spite of his intense interest in shoulder lesions, he has never been able to operate upon a patient with a recent tear.

FIG. 1. Patient attempting to abduct right arm four days after rupture of right supraspinatus tendon.

The reason for reporting this case is to confirm Dr. Codman's predictions that immediate repair of the tendon would produce a practically complete return of shoulder function soon.

The patient, an Irish janitor of sixty-three years, was cleaning a washroom. His feet slipped on the soapy floor and, as he fell on his right side, he raised his right arm to protect himself, thus striking his right elbow. He noticed a sharp pain in the region of the right shoulder immediately after the accident and was seen the following day because of the persistence of pain. On examination he was found to have no swelling or deformity in the region of the shoulder joint, but there was definite tenderness on pressure over the greater tuberosity. All motions of the shoulder were normal and without marked discomfort. He was unable to abduct his arm more than 45° and even this motion seemed to be largely due to rotation of the scapula (Fig. 1). If, however, his arm was passively abducted to the horizontal by the examiner (Fig. 2), the patient could continue abduction until his arm was brought above his head (Fig. 3). This motion was accomplished without marked discomfort. When the patient was asked to stoop over, he could swing his arms without disability and in this position, in which the function of the supraspinatus was not necessary, he could elevate his arms above the head and maintain them thus while he raised his trunk to the erect position (Figs. 4 and 5). When the arms were permitted to descend in abduction there was a decided jog on the right side as they reached the point just a little below the horizontal. At this point the right arm seemed to fall as if unsupported and the patient experienced a slight pain.

A diagnosis of rupture of the supraspinatus tendon was made, based on the following points: (1) history of fall on the elbow with pain in the shoulder; (2) inability to abduct the arm; (3) ability to continue abduction if the arm was passively raised to the horizontal (the supraspinatus which begins abduction could not function but the deltoid could continue abduction after the arm was raised to a point where it was mechanically efficient); (4) ability to elevate the arm above the head while in the stooping position (Codman points out that in the stooping position gravity permits the arm to fall forward without the necessity of a

[1] Codman, E. A. The Shoulder. Boston, 1934.

* From the Surgical Service of Dr. E. L. Eliason, Philadelphia General Hospital.

functioning supraspinatus muscle); (5) the definite jog in the descent of the arm as it passed the point where the supraspinatus normally holds the weight of the arm as it descends to the side.

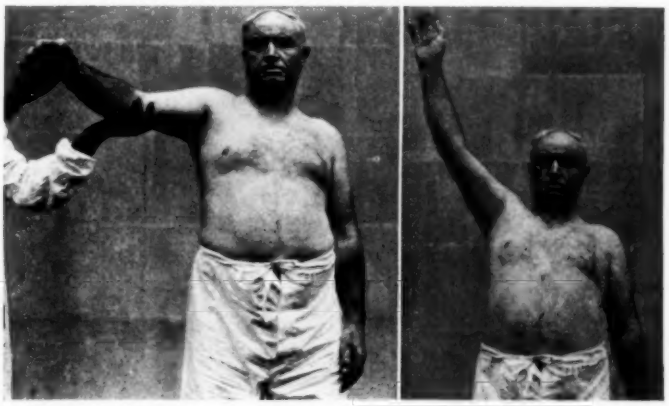

Fig. 2. Fig. 3.

Figs. 2 and 3. If arm was passively abducted to horizontal (Fig. 2), patient could complete abduction (Fig. 3).

An *x*-ray examination of the shoulder failed to reveal any bony lesion or any calcification in the region of the supraspinatus tendon.

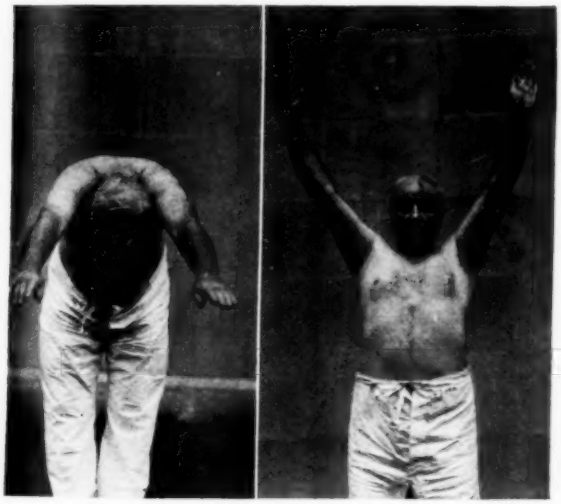

Fig. 4. Fig. 5.

Figs. 4 and 5. If patient leaned forward at waist he could elevate his arms above head because function of supraspinatus is not necessary to perform this motion (Fig. 4). He could then stand erect and maintain his arms in the elevated position (Fig. 5). (Codman's stooping exercises.)

The patient was operated upon on June 26, 1934, four days after the accident. Under brachial plexus block a small incision about 1½ inches long was made over the greater tuberosity with the arm held in internal rotation at the side. After separation of the fibers of the deltoid, the subacromial bursa was opened and a distinct tear of the supraspinatus was encountered. The edges of the tear were widely separated at the anterior portion of the tendon, but the posterior portion was partly held together near the attachment of the infraspinatus muscle. Silk mattress sutures were inserted and the arm abducted as they were tied, pulling the torn edges of the tendon together (Fig. 6). The wound was closed with interrupted sutures in the deltoid and clips in the skin. The arm was dressed with a fairly thick axillary pad so as to hold it in partial abduction. A wrist sling and body swath completed the dressing.

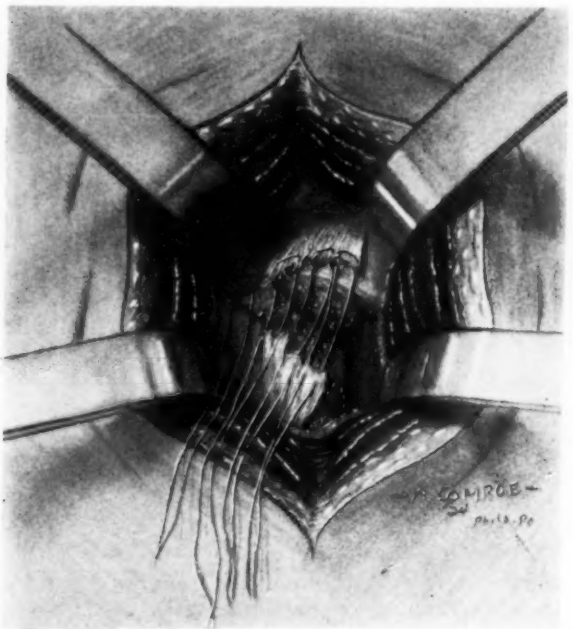

Fig. 6. Sketch of operative field showing separation of deltoid, and tear of supraspinatus tendon after mattress sutures of silk had been inserted.

The patient was allowed out of bed the day following operation and was discharged on the fifth day, after the skin clips were removed. For a period of a week, the patient carried his arm in the sling but at frequent intervals he

performed the stooping exercises, the pendulum-like swinging of the arms while bending forward from the hips. Two weeks after operation, the arm was placed on an abduction splint and the patient was encouraged to release the wrist strap for the purpose of obtaining external rotation of the humerus.

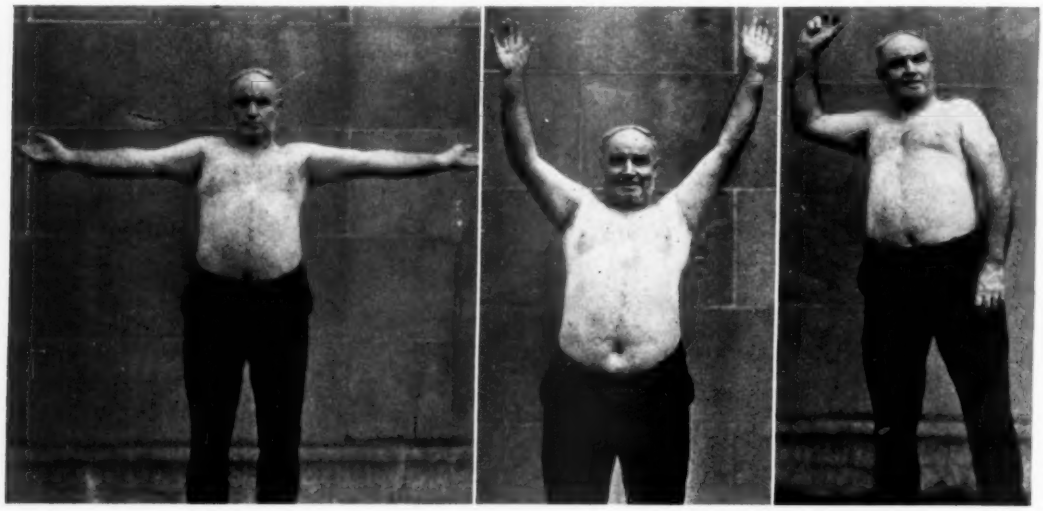

Fig. 7. Fig. 8. Fig. 9.

Figs. 7–9. Patient ten weeks after suture of right supraspinatus tendon. Note slight restriction in extreme abduction, and slight disability in external rotation with arm abducted to horizontal.

After two weeks the splint was removed and the patient was taught to continue his stooping exercises. The motion obtained was somewhat increased by having the patient hold on to a waist-high table while he gradually stepped backwards as far as possible, thus bringing his arms into more and more complete elevation above his head. In addition he was instructed to stand as close as possible to a perpendicular wall and to work his hands upward along the wall. He returned to his work on August 1 and his occupation, sweeping with the broom, was substituted in large part for his exercises so that in a few weeks' time he had a fairly good range of motion without pain.

A follow-up examination was made on September 11, approximately ten weeks after the suture of his tendon. At that time he was performing all the regular duties of his occupation without any disability or discomfort. Motion in the shoulder joint was free and there was no tenderness over the greater tuberosity. The only restriction in motion was in extreme abduction (Fig. 7). He did complain that he "had to throw a ball like a girl" because he couldn't completely externally rotate his arm when it was abducted to the horizontal (Fig. 8).

DISCUSSION

Codman points out that the following conditions, symptoms and signs indicate a complete rupture of the supraspinatus tendon and are present within the first twenty-four hours after injury:

(1) The patient must be a laboring man or woman, (2) age forty to seventy, (3) known to have previously had a useful, painless shoulder, who, (4) at the time of an adequate injury had (5) a sharp pain in his shoulder, (6) followed by a wakeful night; (7) inability to raise his arm; (8) negative *x*-ray; (9) no restriction of motion when stooping; (10) faulty scapulohumeral rhythm (ascending in flexion, descending in extension); (11) a tender point with (12) a sulcus and (13) an eminence, at (14) the insertion of the tendon of the supraspinatus, which causes (15) a jog, (16) a wince and (17) a soft crepitus, as the tuberosity (18) disappears under the acromion when the arm is passively elevated, and usually also as it reappears during descent of the arm.

In this patient no sulcus or eminence was noted at the insertion of the tendon of the supraspinatus and there was no soft crepitus elicited as the tuberosity passed under the acromion when the arm was passively elevated. Otherwise all of the signs were demonstrable. The case is of interest because it shows the very satisfactory result which may be obtained when suture of the torn supraspinatus tendon is performed soon after the rupture.

CUTANEOUS HORN OF THE SCALP*

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THE first case of a cutaneous horn of the scalp was reported in the literature by Lafrancus[1] in 1300, occurring in a man who had "7 swellings of the scalp." The largest was the size of a thumb and resembled a "goat's horn." The outstanding case of a cutaneous horn of the scalp, so often quoted in the literature as the largest horn ever reported (also quoted by Ewing) is that of a Mexican porter, Paul Rodriguez.[2] It measured 14 inches in circumference, and divided itself into three branches a few inches from the root. The man was never seen without a handkerchief covering his head. Only after being struck on the head by a barrel of sugar was he brought to the hospital, where the horn was discovered.

Although some writers believe that cutaneous horns have their origin in warts, the majority of cases reported began as sebaceous cysts, especially cutaneous horns of the scalp, which comprise about 50 per cent of all cases. Individual cases have been reported on the face, eyelid, nose, cheek, lower jaw, ear, thigh, buttock, penis, scrotum, clitoris, and back (spinal region). Although horns in humans resemble in many respects the horns of cattle, they are attached to the scalp and not to the bony structure.

The greater number of cases reported occurred in females. Old age is considered a predisposing cause, although no age is immune. Three cases have been reported in infants. The majority of cases that occurred on the scalp began as sebaceous cysts, which were traumatized or only partially removed by surgery. The contents of the cyst dries up, accumulates layer upon layer in concentric fashion, thus forming the horn. As the horn grows, it tends to curve at the tip by mechanical interference from clothing. The base of the horn has been found to have undergone epitheliomatous changes in about 12 per cent of the cases. Occasionally these horns have been shed or have been knocked off, only to recur.

Of all the writers on the subject, Erasmus Wilson[3] gives the most complete pathological description of cutaneous horns arising from sebaceous cysts. He believes that from the torpid action of the skin or abnormal contents of the cells, or both acting together, the sebaceous substance collects within the follicles, becomes impacted, and acquires an abnormal degree of density. This impacted mass by its pressure obstructs the circulation in the follicles, thus sebaceous substance ceases to be produced, while the formation of epithelium still continues. Layer upon layer of epithelial scales are developed until the mass acquires considerable size.

Dubland and Cruveilhier (quoted by Wilson) made a clinical analysis of human horns and found that the horn is chiefly composed of albumin, a small quantity of mucus, phosphate of lime, chloride of sodium, and a trace of lactate of soda.

Ewing believes that "the persistence of blood vessels in very fine papillae coursing through the horny material accounts for the extensive development of the greatly elongated forms." This is borne out by the fact that the majority of horns that have been removed were found to be very vascular at the base of the horn where they were excised. Wide excision is recommended by most writers. Particular care must be taken when sectioning the horn to examine especially the base for epitheliomatous changes.

No attempt is made here to abstract the various cases of cutaneous horns, as the histories and physical findings are

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more or less alike. The majority of cases began as sebaceous cysts, some originating in senile warts, some in old scars, burns, or "chicken bite" on the hand.

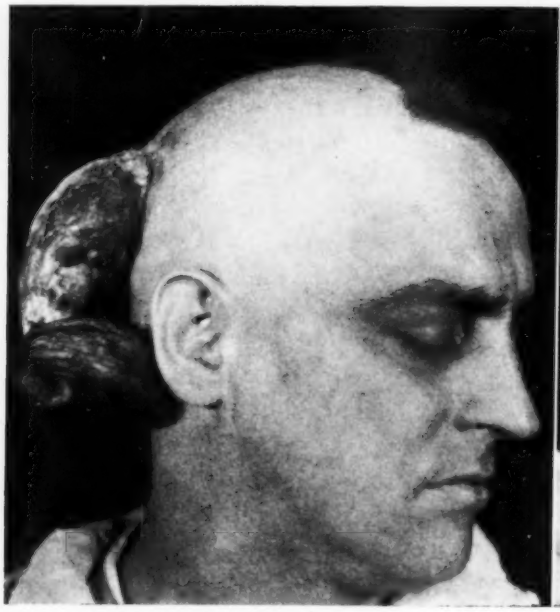

FIG. 1. Anterior view.

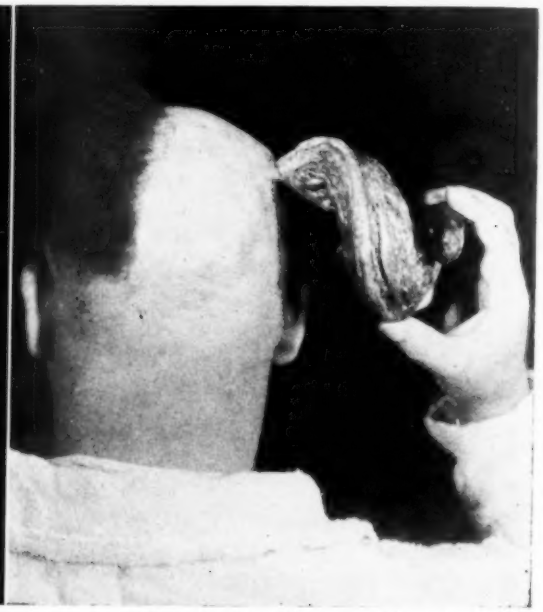

FIG. 2. Attachment of horn.

A white adult male, aged thirty-four, was admitted to Cumberland Hospital on June 17, 1934, complaining of having a horn on his head. About six and a half years before, he was struck on his head during a fight. A month later he noticed a lump had developed in the same area the size of a pea. It was very hard and hurt him when he combed his hair. He went to his doctor, who took it out. The patient states that it looked like a piece of "gristle."

About six months later the growth reappeared. This time the patient refused to have it taken out because the doctor "should have done a good job the first time." However, it began to grow very fast and became very noticeable, so the patient always wore a hat which held the horn to the side. For six years his children never saw their father without a hat, as he did not wish to frighten them. He worked on a furniture truck owned by a large department store in New York City. None of his fellow workers had ever seen him without a hat until one day his hat was blown off by the wind, and they were horrified to see the "devil man." They reported him to their employer, who gave the patient notice that he would have to have the horn removed or lose his job. He chose to lose the job rather than give in to the doctors. However, after being out of work for several months, starvation led him to Cumberland Hospital to have the horn taken off.

The past and family history were essentially negative. He had always been in good health.

Physical examination revealed a young adult male, very well built, in excellent physical condition with the exception of the growth on his scalp, as shown in Figures 1 and 2. Examination of his head revealed a large horny structure, simulating a ram's horn, with two spirals, at the right occipitoparietal region, measuring 20 cm. in length and 8 cm. in diameter at its mid portion. It was attached by a small pedicle (Fig. 2) and hung down the patient's neck as far as the lobe of the ear.

The horn was excised by Dr. Rudolph Goldberg. It was found to be very vascular at the base. The bleeding parts were clamped and ligated, and the scalp was approximated with catgut. Following is the pathological report of Dr. S. H. Polayes: "Microscopical: Tissue consists entirely of concentric layers of horny epithelium. Diagnosis: Hyperkeratosis (horn)."

The patient was last seen at his home on December 14, 1934. There is no evidence of recurrence. The wound healed with a small keloid formation about 0.5 cm. above the surface of the scalp.

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2. *New York Med. Repository*, 20: 88, 1820.
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DERMOID CYST OF THE OVARY

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LARGE cysts of the ovary which were so frequently seen in former years are now seldom seen because of their earlier recognition and also because women have become better educated in such matters. Consequently cysts and other tumor formations are removed before they reach the astounding dimensions of those seen in former years.

Dermoid cysts of the ovary are not rare, however they seldom reach the size of the one reported here.

On March 13, 1934, Mrs. E. A. appeared at my office for examination. Her chief complaint was enlargement of the abdomen which had been present for eleven years, although during the last year the enlargement had been more rapid than at any time previously, and she had not felt as well recently. She said that she hadn't the same strength and endurance as formerly. Considerable difficulty was experienced in performing her usual duties and she spoke of difficulty in breathing. There had never been any pain connected with the enlargement and furthermore there had been no irregularity of menstruation. Menstruation began at the age of thirteen and her periods were considered quite normal. She had been married about twenty years and there had been no pregnancies.

Inventory by system failed to reveal the presence or history of any other abnormality of other organs. There was no history of accidents or of operations.

The physical examination revealed a white female about thirty-five years of age who appeared cachectic and although much out of proportion in size and configuration, nothing unusual could be elicited except the huge enlargement of the abdomen which on percussion revealed the presence of fluid quite unlike that of ascites. The costal margins were flaring and respirations were very shallow and labored.

The laboratory report showed that a marked secondary anemia was present. The R.B.C. numbered 1,960,000 per cubic m.m. with hemoglobin of 45 per cent. There was slight poikilocytosis. The W.B.C. numbered 5500 per c. mm. with 2 per cent myelocytes, 1 per cent juvenile forms, 8 per cent staffs, matured cells 60 per cent. There were 29 per cent lymphocytes and 71 per cent polymorphonuclears. Urine analysis showed albumin one plus as the only abnormality.

The patient was admitted to the hospital on March 14, 1934 and on the following day she was given 500 c.c. of citrated blood intravenously. On the second day the R.B.C. had risen to 3,620,000 with hemoglogin of 55 per cent. On March 17, 1934 she was given another 500 c.c. of citrated blood which raised the R.B.C. to 3,900,000 with hemoglobin of 60 per cent.

On March 19, 1934 she was considered safe for surgery and laparotomy was performed under local anesthesia and nitrous oxide. It was considered inadvisable to attempt to remove this large tumor in toto, because of the shock that sometimes accompanies the rapid decompression of the abdomen. It was reasoned that if the cyst could be tapped and spilling prevented, that gradual decompression of the abdomen could be accomplished with the minimum of shock and risk to the patient. The skin was infiltrated with novocaine of ¼ of 1 per cent and then a small incision made through the skin and fascia down to the cyst wall. A trocar was introduced and sutured in place with a pursestring suture. Seven gallons of chocolate-colored material were gradually withdrawn from the main compartment and spilling was entirely avoided. The removal of this quantity of fluid made the removal of the remaining portion of the cyst a relatively simple matter.

The specimen when removed showed that we were dealing with a cyst that had characteris-

tics of a dermoid. The main compartment contained 7 gallons of fluid with the consistency of chocolate syrup. This fluid was later

Fig. 1.

proved by laboratory tests to be old blood.

The second compartment contained about a pint of oily substance, evidently sebaceous material. The third compartment contained a lock of hair and a tooth in a cartilaginous base.

The left ovary was enlarged to about the size and shape of a tangerine, and had characteristics resembling the larger tumor although there were no dermoid characteristics. It was also removed. The appendix was normal. The uterus and tubes were also normal in size and consistency.

The following pathological report by Dr. J. P. Simonds of Northwestern University Medical School is submitted.

"The specimen is a huge multilocular cyst of the ovary which has collapsed as a result of escape of the fluid through incisions in its walls. The largest cyst easily measures (after fixation) about 25 cm. in diameter. Its wall measures from 0.5 to 1.5 cm. in thickness and its lining is smooth. A second cavity is 12 cm. in thickness. This cyst is filled with a mass of yellowish soft greasy material containing an abundance of curly brown hair. From one place in the wall there projects a flat, sharp edged tooth 11 mm. in length.

"Sections of the wall of the largest cyst show along the outer margin, a large amount of compressed ovarian tissue. Inside this is a layer of fibrous tissue. No epithelial lining is present in these sections.

"Diagnosis: Simple multilocolar cyst and dermoid cyst of the ovary."

The postoperative course was uneventful and the patient was discharged on the eighteenth postoperative day. At the present writing the patient is perfectly well except for a few hot flashes which are readily relieved by the daily administration of 3 gr. each of whole fresh ovary and pituitary substance.

GONOCOCCAL PERITONITIS BEFORE PUBERTY

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GONOCOCCAL infection of the peritoneal cavity of young girls before puberty is a rare disease, as recently emphasized by Vest.[1] This report of a case, operated on by the author, will include a brief summary of the major points emphasized by Vest in his paper, rather than repeating his survey of the literature.

The venereal manifestations of gonorrheal infection in adults have been recognized from antiquity. The bacteriologic cause of the disease, however, was not recognized until Neisser discovered the intracellular diplococcus in 1879. The clinical picture of acute peritonitis from infection by the gonococcus was first suggested by West in 1858. Case reports which were probably true instances of gonococcal peritonitis, although not verified by bacteriologic studies, were reported by Saenger, Lowen and Hatfield in 1886. Shortly afterwards case reports by Penrose, Huber, Stevens and Menge were presented, although these were not confirmed by bacteriologic studies. In 1895 Frank, and in 1897 Mejia presented 2 cases of fatal peritonitis in which cultures from the peritoneal cavity showed the gonococcus. In 1897 Cushing reviewed the literature on the subject and reported a case of general peritonitis operated upon by him and from which Young developed cultures of the gonococcus. The next case so proved was reported by Hunner in 1902. Vest himself reported 2 cases with bacteriologic confirmation. In 1891 Wertheim produced acute gonococcal peritonitis experimentally by injecting the gonococcus into the peritoneal cavity of various animals.

Vest collected reports from the literature of 51 cases of gonococcal peritonitis occurring before puberty. The ages of the infected children varied from three months to twelve years. Only 10 of all these cases were completely proved by demonstrating the gonococcus in smears and cultures taken from the peritoneal cavity. The remaining 41 cases were diagnosed on the basis of the clinical picture of general peritonitis and an associated gonococcal vulvovaginitis, without any other demonstrable intra-abdominal infection (i.e. appendicitis). The case reported in this paper was of an acute peritonitis associated with a vulvovaginitis having a profuse discharge, the smears of which showed the gonococcus.

There are two theories held regarding the etiology of gonococcal peritonitis before puberty. Morris believes it must develop secondary to a gonococcal septicemia, since the mucous plug blocking the cervix before menstrual life would prevent infection spreading up from the genital organs. Others hold that the infection must be the result of direct extension from the pelvic organs, since arthritis and other complications of gonococcal septicemia are strikingly absent from the clinical picture. Such a septicemia they maintain would exhibit unusual selectivity to infect only the peritoneal cavity.

Vest states regarding the pathology of the condition:

> There has been some variation in the pathologic findings at autopsy or operation. In general two types are described. In the fibrinous or plastic type there is very little free pus in the peritoneal cavity. The intes-

[1] Vest, S. A. Jr. Generalized gonococcal peritonitis in children. *Intenat. S. Digest*, 16: 3–12 (July) 1933.

tines, parietal peritoneum and omentum are injected with considerable fibrin deposits throughout the abdominal cavity. The fibrin can be removed as small flakes and sometimes as large cakes. Little or no purulent exudate is present. In some cases adhesions are beginning to form. In the second type, especially found in children, there is considerable odorless purulent exudate. It may be thin and white, but some cases have an exudate which is viscid, glairy material with a dirty color or appearance. Early adhesions are rare in this type. The tubes may be inflamed and swollen and pus can usually be expressed from the fimbriated ends. What accounts for the excess of pus in one case and fibrin in the other is not clear. Both types are found in adults and in children and in cases in which secondary infection is not a factor.

There are two clinical types of cases: acute and chronic. They differ chiefly in that the acute cases last from five to ten days, while the chronic cases exhibit fever and peritoneal irritation for two or three weeks. Both types have a characteristic abrupt onset, with fever and signs of peritoneal irritation. The average temperature is 102°, but may reach 104° to 105°F. The abdomen may be moderately painful to palpation, or extremely sensitive to the slightest pressure. Usually the tenderness is generalized and not localized at McBurney's point. Boardlike rigidity is absent. In very severe cases there is a picture of septic peritonitis with prostration, pale drawn facies, rapid thready pulse, cold extremities and persistent vomiting. The abdomen may or may not show moderate distention.

Comby and the French writers were the first to demonstrate that operative intervention in these cases is not necessary. In 1901 Comby reported 8 cases which were not operated upon, but were treated with the usual routine of fluids, sedatives, Fowler's position, care of distention, treatment of vaginitis, and the use of vaccines.

In differential diagnosis one must consider peritonitis of appendiceal or pneumococcal origin. Vest states that, "If any doubt as to the diagnosis exists the risk of exploration is far less than the danger of failing to recognize acute appendicitis." The mortality of all reported cases was high, 31 per cent. In the unoperated group it was 30.5 per cent, while in the operated group it was 33 per cent. Operative intervention did not seem to have affected the mortality rate. The present patient was operated upon and survived. The diagnosis was based upon the operative findings of an idiopathic peritonitis in the presence of an active vulvovaginitis from which the gonococcus was recovered.

HISTORY

The patient, a girl aged ten years, was admitted to the hospital at nine o'clock in the evening because of an attack of acute abdominal pain of twelve hours' duration.

The mother and father were living and well. One sister died from cause unknown. In childhood the patient had chicken pox, measles and mumps.

The mother said that for a week preceding the onset of the present attack of abdominal pain, the child had been having marked pain and burning on urination, with a foul yellowish vaginal and urethral discharge, enough to stain the clothing.

On the morning of admission, a week following the onset of the vaginal discharge, the patient was seized with lower abdominal pain, enough to double her up. The pain was localized chiefly in the right lower quadrant. With the onset of the pain the vaginal discharge seemed to increase. During the day the patient vomited six or seven times. On admission to the hospital she still complained of pain in the right lower abdomen.

Examination: The patient lay quietly in bed appearing acutely ill. There was an apprehensive anxious expression on her face with pinched hippocratic facies. Examination of the head, throat, heart and lungs was negative. The abdomen was slightly but definitely distended. There was generalized tenderness in the lower half of the abdomen, most marked at McBurney's point. There was rigidity of the lower right rectus muscle, partly voluntary, but more marked than on the left side. The temperature was 101.8°F., the pulse 120, and the respirations 20.

Laboratory: The white blood cell count was 20,150 with 92 per cent polymorphonuclear leukocytes, 7 per cent small lymphocytes and 1 per cent eosinophiles. The urine was negative except for the slightest possible trace of albumin and clumps of white blood cells. A vaginal smear made before operation (not done by the laboratory) was reported erroneously as showing gram positive intracellular diplococci. Repeated vaginal smears taken after operation were correctly reported by the laboratory as showing gram negative intracellular biscuit-shaped diplococci: the gonococcus.

Preoperative Note: The patient was well prior to the day of admission except for a vaginal discharge of one week duration. She had acute severe right lower abdominal pain since morning, and appeared acutely ill on admission. The symptoms were too severe and the patient appeared too prostrate for the twelve hour period of illness, to feel certain that the diagnosis was appendicitis. The differential diagnosis of gonococcal or pneumococcal peritonitis was considered with a presumptive diagnosis of pneumococcal peritonitis because of the erroneous report of gram positive diplococci in the smear. However, because of the marked rigidity and tenderness in the right lower abdomen, it was felt inadvisable to leave the child without exploring the appendix.

Operation: A low right rectus incision was made, reflecting inwards the outer border of the rectus muscle. The peritoneum was injected as was the serosal covering of all the intestines. There was a moderate amount of tenuous, somewhat dirty glairy exudate in the lower abdomen. The appendix itself had a serositis similar to that of the bowel, but was not of itself acutely inflamed. A single culture from the peritoneal cavity was taken on the assumption that it was a pneumococcus peritonitis but no growth was procured on agar. The appendix was removed, and the abdomen closed without drainage. The patient made an uneventful convalescence. The temperature was normal on the second day. The incision healed without suppuration. The vaginitis was treated locally and was fairly well cleared up when the patient was discharged from the hospital.

Postoperative Note: Subsequent investigation of the child's environment brought to light a probable source of infection. For obvious reasons the child was not questioned directly about sexual exposure, but the mother was interviewed. The patient, she said, had an aunt about the same age as the patient herself, with whom she slept. One day the mother noticed that the aunt who had gone to the bathroom with a neighbor's child was staying there longer than seemed natural. The mother went to the bathroom and found the two children rolling on the floor in close embrace. Both of the children, according to the mother, had a vaginal discharge. It is highly probable that the patient sleeping with the aunt was infected by her. The aunt was examined at the hospital, but the vaginitis had cleared, and no gonococci were found in the smears.

SUMMARY

The local manifestations of gonorrhea as due to infection by a specific organism has been recognized since 1879. But the clinical picture of peritonitis as due to invasion of the peritoneal cavity by the gonococcus was not proved by culture of the organism until 1895. Gonococcal peritonitis in young girls before puberty is rare. The earliest cases reported were diagnosed on the basis of the clinical picture of peritonitis, with an associated vulvovaginitis. Later cases were reported in which the organism was recovered directly from the peritoneal cavity by smears, and cultures in ascitic fluid. Of the 51 cases collected from the literature by Vest, only 10 were completely proved.

The clinical picture is of an acute peritonitis associated with a purulent vulvovaginitis from which the gonococcus can be recovered.

The general mortality rate of gonorrheal peritonitis before puberty is 31 per cent. Of the cases in the literature the mortality without operation was 30.5 per cent and with operation 33 per cent, so exploratory laparotomy did not seem to affect the mortality rate appreciably. "If any doubt as to the diagnosis exists, the risk of exploration is far less than the danger of failing to recognize acute appendicitis." On this basis many cases, including the author's, have been operated upon. This report brings the number of cases in the literature of gonorrheal peritonitis before puberty to 54.

UMBILICAL HERNIA WITH UNUSUAL CONTENTS

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SPRINGFIELD, O.

MANY cases of umbilical hernia with variable hernial contents have been presented, but a perusal of the literature does not reveal any with such unusual contents, which is my reason for the publication of this case.

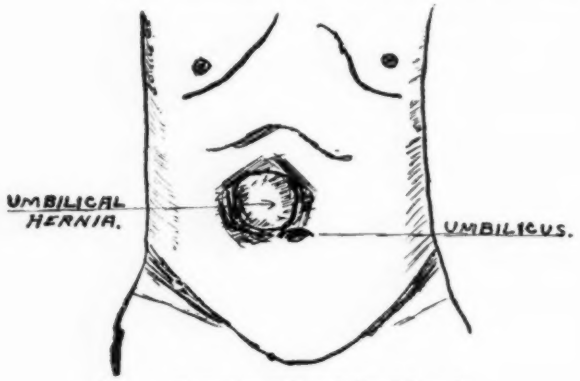

FIG. 1. Location of umbilical hernia.

The hernial contents consisted of the gall bladder and two supernumerary lobes of the liver, suspended from the quadrate lobe by a hinge-like pedicle of liver tissue.

R. LeV., a white male, aged fifty years, was admitted to the Springfield City Hospital, complaining of acute pain in the epigastric region of the abdomen. The patient gave a history of vague abdominal pain for a period of five years. The pain was not constant but occurred in the form of exacerbation, it was always associated with eating and would appear one and one-half to two hours after taking food. The pain was of a gnawing character and was often relieved by taking bicarbonate of soda. This complaint has never been severe until a few days ago. The patient associated the complaint with a mass which presents itself on the abdomen just above the umbilicus and to the right of the midline. He did not recall the duration of the mass.

The previous evening, the abdominal pain became more intense and the mass increased in size. He became nauseated and vomited. The vomitus as he described it was only of gastric origin. His bowels moved regularly up to the acute onset, but have been irregular since.

Physical Examination. The patient was a well-developed and apparently well-nourished male, lying in bed on his back and very restless, changing his position often. He complained of an intense headache and abdominal pain. His face was weather beaten and had a peculiar bluish tinge which was not cyanosis, being confined largely to the forehead and cheeks. He was cooperative, but appeared to be in acute distress. Head, nose, ears and throat were normal.

Eyes. Pupils were of moderate size and reacted to light and accommodation. Internal strabismus of the left eye, conjunctiva injected, sclerae discolored but not icteric.

Mouth. Teeth in poor condition. Pyorrhetic pockets numerous. Buccal mucous membrane normal.

Chest. Antero-posterior diameter was increased. Evidence of childhood rickets present. Chest moved little with respiration. The respiratory movements were mainly abdominal. Tactile fremitus was diminished. Percussion note was somewhat hyper-resonant. Breath sounds were distant, but apparently normal.

Heart. Not enlarged to percussion. No shocks or thrills, apex beat, fifth left interspace, mammary line. Heart sounds were distant, regular in rate and rhythm. No murmurs. Peripheral vessels had thickened walls. Pulses were equal and of good tension. Blood pressure 160/90.

Abdomen. Below chest wall, moved with respiration. No scars. There was a soft mass, about 2.5 cm. in elevation and measuring 6.5 × 4 cm., above the umbilicus 1 cm. and to the right of the midline. (Fig. 1.) The mass was soft and gave rise to a gurgling sensation on palpation. The mass could be reduced in size by manipulation, a spherical opening could be palpated in the rectus muscle, the opening would admit two finger tips easily. When the patient coughed the mass protruded and there was no doubt as to the presence of a loop of intestine. The mass when of the maximum size was quite tender to palpation. There were no other masses present in the abdomen. There was tenderness in the epigastric region, but no rigidity.

Genitalia. Large left hydrocele. Testicles appeared normal, but the left epididymis was enlarged and slightly tender.

Extremities. Normal. Reflexes normal.

Temperature 99°F., pulse 70, respirations 22. Hb. 90, R.B.C. 4,400,000; leucocytes 8400;

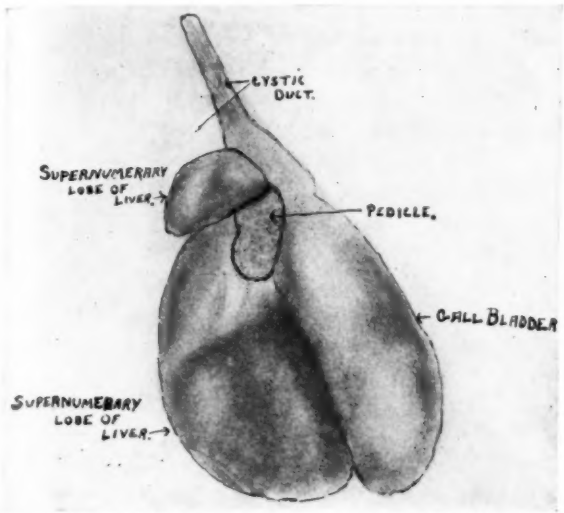

Fig. 2. Gall bladder, two supernumerary lobes of liver, pedicle and cystic duct. (Hernial contents.)

neutrophiles 81 per cent; small lymphocytes 18 per cent; large lymphocytes 1 per cent. Urine negative.

Diagnosis. Umbilical hernia (acquired). Left hydrocele. Dental caries. Pyorrhoea alveolaris.

Operation. A crescentic incision was made above the umbilicus, slightly to the right of the midline. On opening the sac, the gall bladder and supernumerary lobes of the liver presented in the incision. At the lower inner margin of the hernial ring was attached the neck of the gall bladder by a narrow adhesion. The incision was enlarged to the right and left to afford adequate exploration. Exposure revealed in the liver the fissure where the gall bladder is usually located, the elongated cystic duct and the small supernumerary lobe. The supernumerary lobes of the liver with the gall bladder attached were the only hernial contents. The cystic duct was fully 15 cm. in length, the gall bladder of normal size, thin wall, normal color, and not containing any stones. (Fig. 2.) The supernumerary lobes were attached by a pedicle of liver tissue to the quadrate lobe of the liver. (Fig. 3.) The lobes were removed at the pedicle portion with the gall bladder. Hemostasis of the pedicle stump was controlled by suturing with catgut. Repair of hernial opening was by a transverse overlapping of aponeurotic flaps as described by Mayo.

At a later date, patient had a second operation, for correction of the left hydrocele (Beck's bottle operation) and at the same time the extraction of twenty-four teeth.

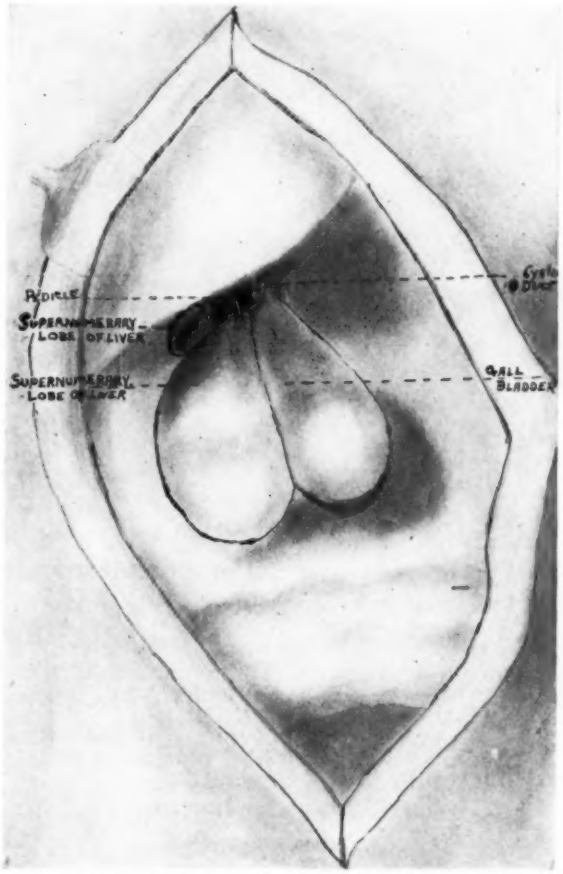

Fig. 3. Gall bladder and supernumerary lobes, suspended from quadrate lobe of liver.

Pathological Report. The specimen consisted of the gall bladder and two supernumerary lobes of the liver. The supernumerary lobes were attached to quadrate lobe of the liver by a pedicle measuring 1.5 × 1 cm. The larger lobe measured 6 × 4.5 × 3 cm. and the smaller lobe 3.5 × 2 × 1 cm. The gall bladder was attached along one margin of the larger lobe and was freely movable with this portion of the liver, possessing an unusually long, cystic duct. The gall bladder measured 12 × 3 cm. and presented no noteworthy pathology.

No microscopical sections were taken, the tissue being preserved intact as a museum specimen.

Diagnosis. Gall bladder attached to two supernumerary lobes of the liver.

Postoperative Notes. The patient had an uneventful convalescence and was discharged from the hospital four weeks following his admission. He has remained in good health and has not had any return of symptoms.

STRANGULATED INTERNAL HERNIA SIMULATING APPENDICITIS*

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AND

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CHICAGO

MANY pathological processes in and about the ileocecal region are obviously diagnosed appendicitis. In a fair percentage of cases the only excuse for such a diagnosis is topographical, as a thorough clinical appraisal of the sequence and the degree of symptoms will often make a diagnosis of appendicitis. There are, however, a number of entities in McBurney's zone which will tax the acumen and experience of even seasoned clinicians.

Internal strangulated hernias about the ileocecal region are invariably diagnosed acute appendicitis, especially those which become strangulated in one of the several pericecal and pericolic fossae. Steinke,[1] reviewing the literature of internal hernias between the years of 1925 and 1932, cites 7 cases of strangulated pericecal hernias, the strangulation being either in one of the fossae or in a mesentery slit, all of which were diagnosed appendicitis. Pribram[2] cites the case of a man fifty years old who suffered from chronic constipation and had at various times paroxysms of abdominal pain and vomiting. One day he had a flare-up of abdominal pain in the right ileac fossa associated with vomiting, which assumed the picture of appendicitis. Exploratory laparotomy revealed 235 cm. of strangulated, gangrenous small intestine in a retrocecal fossa. The appendix was grossly normal. The gangrenous segment was resected and the patient made a perfect recovery. Traum[3] reports a case of a patient who had never been ill previously and who was suddenly seized with abdominal pain in the right ileac fossa, vomiting and inability to defecate or pass flatus. A diagnosis of appendicitis was made. (We feel that the sudden pain in the right ileac fossa, from the very start, associated with inability to pass flatus or defecate should have suggested intestinal obstruction).

Occasionally the appendix is a part of the pathology, as in the case reported by Zoepfel.[4] The patient, a male eighteen years of age, had an incarceration of the entire small intestine in a retrocecal recess. The appendix was perforated and was a part of the pathology.

Several cases were reported by Muller,[5] Stech,[6] Malcomb,[7] Deaver and Burden,[8] Martzloff,[9] and Vidgoff and Sturgeon,[10] in which a preoperative diagnosis of acute appendicitis was made, but upon exploration proved to be strangulated internal hernias.

Coley and Hoguet[11] came closer than many other clinicians to the correct preoperative diagnosis. A case reported by them was diagnosed retroperitoneal hernia before the operation, which proved to be a hernia in the retrocecal fossa.

* From the surgical service of Dr. V. L. Schrager, Cook County Hospital, Chicago, Illinois. Read before the meeting of The Chicago Surgical Society.

CASE REPORT

The patient, a colored male, thirty-three years of age, a cab driver, was admitted to the Cook County Hospital on February 11, 1933, with the examining room diagnosis of acute appendicitis.

In the ward, the interne obtained the following history:

The patient was entirely well until the day of entrance, when about 9:30 P.M. he was seized with a sharp, cramplike pain in the lower quadrant followed by a desire to move his bowels. Driving his cab intensified the pain, and he was compelled to go home. The severe abdominal pains recurred every few minutes, while the pain remained local at all times. Nausea was entirely absent. At home he took sal hepatica three times, each dose being followed by vomiting. He was able to pass gas but not able to move his bowels. At 5 A.M. he was admitted to the Cook County Hospital.

There was nothing relevant in the past history that had any bearing upon the present condition.

Physical examination revealed a colored male, who did not appear ill. Temperature 99°F., pulse rate about 70 and respiration 18. Examination of chest was essentially negative.

The abdomen was moderately distended. There was rigidity and localized tenderness in the right ileac fossa, and there was a palpable mass which was distinctly noticeable on inspection.

The urinalysis was negative in all respects. The leucocyte count was 15,000 with a high polymorphonuclear count. The junior interne made a diagnosis of acute appendicitis, and the patient was referred for surgery.

Operation. McBurney's incision was made. Upon opening the peritoneum, a mass was palpated which adhered to the parietal peritoneum and was located midway between the anterior superior spine and the spine of the pubis. The mass was readily brought up into view, revealing a loop of small intestine of dark red hue contrasting with the normal bowel. The mesentery of the involved bowel showed extensive vascular damage. The freed portion of the bowel became viable, in a short time, and was returned to the peritoneal cavity without resection. On the medial aspect of the cecum there was a rent in the serosa, 1½ inches, which constituted the hernial pocket and readily admitted two fingers. The edges of the rent were indurated and inflamed.

The pathologic picture was that of an internal strangulated hernia, a loop of small intestine being tightly engaged into a rent in the wall of the cecum. (Fig. 1.)

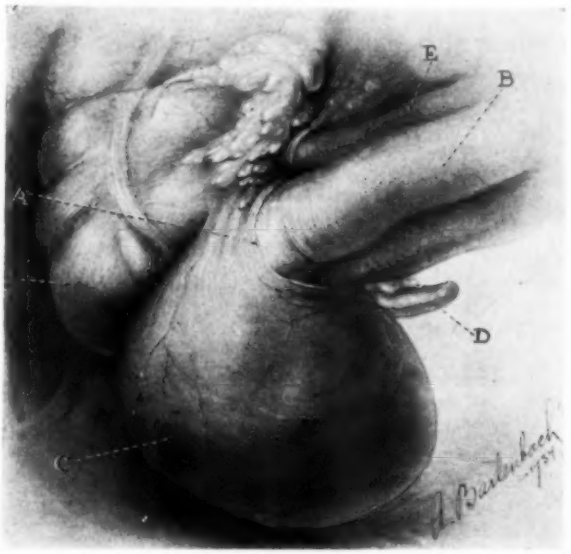

FIG. 1. Artist's drawing of hernia as seen in abdominal cavity. A, Opening into hernial sac. B, Proximal loop of ileum as it enters hernial sac. C, Hernial sac. D, Appendix. E, Ileum as it enters cecum. F, Cecum.

The appendix was grossly normal and was removed routinely.

The patient made an uneventful recovery and left the hospital on the ninth postoperative day.

SUMMARY COMMENTS

Strangulation or incarceration of internal hernias takes place either in the superior or the inferior ileocecal fossa, the retrocolic fossa, or in the ileoappendicular fossa. These hernias occur most commonly in the retrocecal fossa, as shown in the history reports by Short,[12] in which 17 cases were retrocecal as compared with 5 ileoappendicular hernias. Some less common types of strangulations have been reported by Traum,[3] in which the strangulation took place in one of the recesses occassionally found in the ascending or descending colon. In his case, there was no evidence of inflammation at the mouth of the sac. In our case, however, there were distinct inflammatory changes in the

serosa of the cecum. By way of conjecture, we believe that the sac in our case was traumatic in origin, since the patient had no previous evidence of obstruction, although the possibility of a congenital sac, occurring in that particular area, cannot be ruled out.

We are tempted to report this case, because it falls in the common group of internal strangulated hernias in the right ileac fossa, usually diagnosed appendicitis. However, if the clinician would take a painstaking inventory of the quality and the sequence of the symptoms, as stressed by John B. Murphy[13] in his classic article, "Two Thousand Cases of Appendicitis," he would invariably come closer to an accurate diagnosis.

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*Continued from p. 207.

RUPTURE OF THE BLADDER

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As rupture of the bladder cases are very infrequent and as this is a rather unusual injury I feel that it should be reported.

On June 18, 1934 a boy at one of the C.C.C. Camps, near Huntingdon, Pa. was working on an embankment and slipped falling astride of the handle of a shovel, which had been placed against the embankment.

The handle of the shovel struck him just posterior to the anus, then entered the rectum, for a distance of about 18 inches. The shovel handle was removed and he was admitted to the J. C. Blair Memorial Hospital about one P.M.

Condition on Admittance. He was in severe shock, pulse rapid, skin fairly good color. Pupils equal and re-act to light, no discharge from nose or ears, throat clear. The heart is rapid (140) no murmurs. Chest apparently clear. Abdominal examination: the abdomen is rigid over the entire surface and he complains of pain on pressure at all points over the abdomen. He has a desire to void, but did not pass any urine. There was no discharge of blood from the urethra.

He was catheterized carefully, but no urine was obtained, only a small amount of blood. At the anus there is a tear posterior to the anus which extends up into the rectum, opening in the anterior portion of the rectum, about 2 to 3 inches above the anal opening.

Operation, two hours after admission to the hospital. Gas oxygen anesthesia. A midline incision. On opening the peritoneum it was found filled with thin bloody urine. About one quart of this was removed by suction. After carefully walling off the intestines a thorough examination of the pelvis was made. No injury to the upper rectum or sigmoid that was intraperitoneal could be found. There was a wound of the fundus of the bladder, which was about one inch in length. The bladder was cleansed by suction and fecal material was removed from the bladder. The wound in the fundus of the bladder was enlarged and on thorough exploration it was found that at the base of the bladder or near the trigone there was a ragged opening, which was about 1½ inches long. This opening was apparently just between the openings of the ureters. The bladder was thoroughly cleansed and the wound in the lower part of the bladder was sutured with fine catgut. The opening in the fundus of the bladder was then made extraperitoneal and a drain placed down into the bladder. A drain was also placed down into the pelvis and the wound closed by layers to the exit of these drains. A catheter was then placed in through the urethra and also a drain up through the anus and rectum to the base of the bladder.

The postoperative diagnosis was rupture of the rectum, bladder with peritonitis.

Postoperative Course. Immediately following the operation the patient's general condition was fair, but in twenty-four hours he had considerable distention, which was relieved by suction through a small tube placed through the nose into the stomach. His general condition improved and after about one week a small amount of mercurochrome was placed into the bladder and it was found that there was a slight leakage of urine from the bladder through the rectum. This discharge stopped about July 15. On July 25 this boy was transferred to The Walter Reed General Hospital, at Washington, D.C. for convalescence. The following notes were very kindly sent from The Walter Reed Hospital, to be included in this report.

On admission to the hospital his weight was 95 pounds. The midline incision had almost entirely healed except a small tract, which connected with the bladder.

Cystoscopic examination August 6, 1934. Defect at vault at site of suprapubic drainage; moderate defect posterior to trigone; trigone thickened and elevated; no injury to the ureters.

X-ray Findings: Cystogram showed the urinary bladder and urethra are visualized. There is no demonstrable pathology. There is slight residual urine in the bladder after emptying.

August 4, 1934. Barium enema showed distal colon fills well and there is no evidence of pathology. The rest of the colon was negative.

August 8, 1934. Urogram: The right kidney, pelvis and ureter are visualized. The superior and middle calices appear some what blunted. The left kidney and ureter are faintly outlined. The urinary bladder is visualized. There is no demonstrable pathology.

August 14 ,1934. The right urogram negative.

August 18, 1934. Suprapubic wound was healed.

August 30, 1934. He has gained 24 pounds in weight and was very much stronger.

September 23, 1934. Patient has gained 32 pounds. Suprapubic wound healed. There is no incontinence of urine or feces, the bladder emptied well, the sphincter having good control. There was a slight relaxation of the external sphincter and muscle.

On October 3, 1934. I received a letter from this boy stating that he was back at camp and able to do his regular work.

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* Continued from p. 248.

SOLITARY CYST OF THE KIDNEY

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SOLITARY cysts of the kidney are comparatively rare. At the present date of the proved causation has not been found though many theories, some quite fantastic, have been offered.

Reported cases indicate solitary kidney cysts occur most frequently in individuals between the fourth and fifth decades. A cyst is usually located in one or other of the kidney poles and practically never in the central portion.

This case of solitary renal cyst followed none of the usual rules. It occurred in a girl, age ten years, and occupied the middle portion of the right kidney.

The past history of the little girl (C. D.) is negative except for a questionable story of a blow in the right side four years ago, questionable because the father says she was injured and the mother says she was not. The child has been normal as to growth and habits. Mentally and physically she has developed as other children.

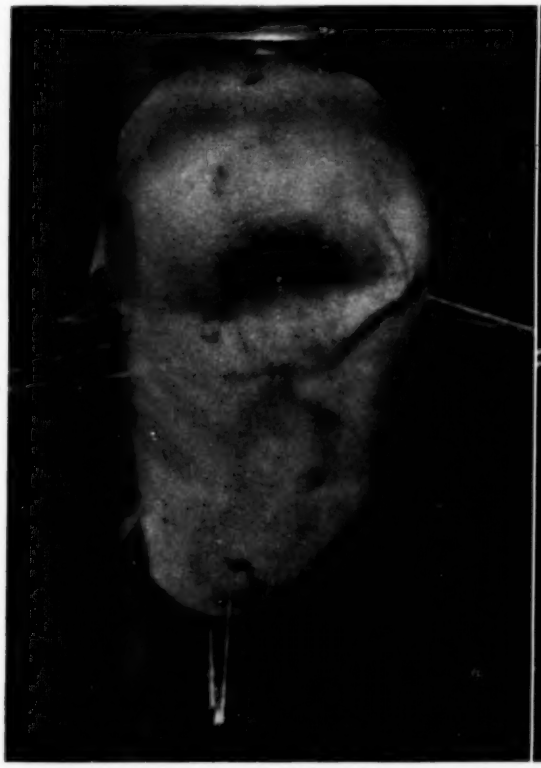

FIG. 1. Peritoneal surface of right kidney showing collapsed cyst wall.

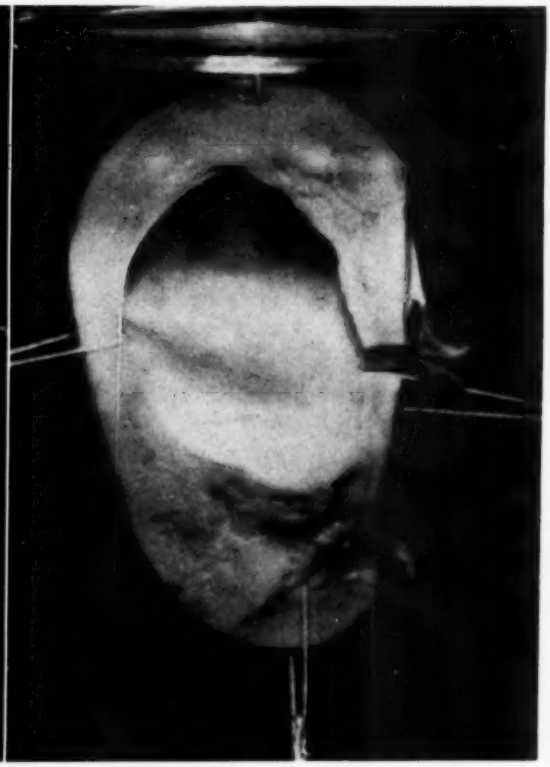

FIG. 2. Bisected kidney looking toward peritoneal cavity. Note size of cyst cavity in relation to kidney.

About three weeks previous to my examination she had been seen by the family physician because of an intestinal upset. This doctor, in the course of his examination, found a mass in her right side. It was not tender and could be moved slightly. When I examined the patient she stated that the mass was tender and had grown noticeably in the past three weeks. Both of these statements were probably

exaggerations due to concentration of attention on the tumor and to apprehension. The mass was smooth, round, and about the size of a small apple. It lay slightly below the ribs to the right of the midline. There was a slight mobility in a vertical direction but none from side to side. The colon lay to the inside and the mass gave the impression of disappearing beneath the liver. Urinalysis reported negative albumin, forty pus cells per high power field, and no red blood cells. The K. U. B. reading was that of a probable tumor involving the right kidney. A right pyelogram showed a compression of the middle calyx with some rotation of upper pole. No cyst could be outlined on the *x*-ray film. The left kidney was normal. Blood count normal. Skin tests for tuberculosis and Wasserman reaction were negative.

Preoperatively we were certain the mass involved to an undetermined extent the right kidney. The rarity of cysts in children of ten years inclined us to rather suspect a ptotic kidney bound in abnormal position by adhesions. In any event, exploration of the right kidney was indicated and this recommendation was accepted.

At operation a rather low placed right kidney was exposed with a large serous cyst in the middle two quarters of the kidney. The large protrusion of the cyst striking the posterior surface of the liver displaced the kidney from its natural bed. The nephrectomy offered no technical difficulties. The patient's convalescence was uneventful and she is now well and strong having gained 12 pounds in weight.

The *pathological report* by Dr. J. A. McIntosh is: "Gross pathology: The kidney measures $9 \times 6 \times 6$ cm. The central portion is bulging and fluctuant. On section, colorless fluid escaped from a smooth lined cavity occupying the central part of the kidney to 50 per cent of its substance. The kidney cortex is entirely absent in an area of 3.5 cm. The cyst cavity is not connected with the pelvis and has flattened the central portion. The lower and upper poles are uninvolved in any way. The kidney substance appears normal except immediately adjacent to the capsule where it is thin and hypoplastic. The capsule strips easily over the uninvolved kidney substance. The kidney pelvis shows petechial hemorrhages. These could have come from rotation of the kidney before amputation. There is no free pus. The cyst contained 30 c.c. of colorless fluid. Chemically it is negative for urea (nitric acid test).

"Microscopic pathology: Section of the kidney pelvis shows submucosal edema with hemorrhages beneath. Section through cyst wall and adjacent kidney cortex shows pressure atrophy and slight tubular dilatation. The glomeruli are normal. The cyst lining is a thin fibrous membrane with flat lining cells. The secreting units appear normal.

"Diagnosis: Unilocular renal cyst."

TUBERCULOSIS OF THE MOUTH*

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IN the war against cancer, the realization that in early diagnosis and treatment lies our only hope for cure has made the examining physician regard with suspicion that group of casual symptoms which may possibly signify cancer. Prominent among these is the presence of a persistent ulceration in the mouth. This cancer suspicion is amply justified because cancer of the mouth accounts for 20 per cent of all deaths from cancer, and is by far the commonest cause of chronic ulceration of the mouth.

Such cases, upon discovery by the dentist or physician, are usually treated for cancer even before the diagnosis has been confirmed by biopsy. In the anxiety to control cancer, another disease which may produce a lesion clinically similar to cancer, but whose treatment and prognosis is totally different, may fail of consideration. This disease is tuberculosis.

Carmody, in 1915, collected from the literature 534 cases of oral tuberculosis. Because of the vigorous public health campaign being waged against tuberculosis today, tuberculosis of the mouth is not a common condition.

Tuberculosis of the mouth is almost always secondary to pulmonary tuberculosis. Carmody described 17 cases in his paper, all of which had definite histories of pulmonary lesions. Levy described 8 cases, all of which had known lung involvement of from one to fifteen years.

The difficulty in diagnosis arises in those cases in which the patient has no knowledge and gives no clear history of pulmonary involvement.

Ivy and Appleton reported 3 cases of oral tuberculosis in which the patient gave no history of lung tuberculosis. The discovery of a similar group of cases in our clinic has prompted the writing of this paper. These 6 cases were originally referred as carcinomata of the mouth. None gave a history of pulmonary tuberculosis. In addition to these cases, this report includes 4 cases of oral tuberculosis in which tuberculosis elsewhere in the body was known to exist previous to the patient's first visits to the clinic.

In the examination of these cases, several points of diagnostic value were noted: the superficial nature of the ulceration, the multiple foci, and the pin-head, yellowish spots have been described by other investigators. In addition, we have noted that the ulceration was usually more irregular than in carcinoma, with small, tongue-like areas radiating out into the surrounding tissue. The surrounding area showed a peculiar puffiness, which on palpation felt soft in contrast to the hard induration of carcinoma. This absence of hardness was striking in a lesion seemingly malignant. The ulceration itself was often crossed by fissures, a finding usually absent in carcinoma. The area might, or might not, be tender, and enlargement of the regional lymph nodes, if present, was due to secondary infection.

CASE 1. J. C., white male, aged thirty-eight, laborer, entered the hospital in August, 1931, comp aining of a swollen, tender lower lip of five years' duration. The lesion began as a small "cold sore" and since then had slowly, progressively spread over the entire lower lip. A Wassermann test at that time was reported 4 plus, but antiluetic treatment failed to give

* From the Radiation Therapy Service, Bellevue Hospital, Ira I. Kaplan, M.D., Director.

any improvement, although it did cause the Wassermann to become negative. Because the lesion persisted, the patient was referred to the Radiation Therapy Department with a tentative diagnosis of carcinoma of the lip. Examination at this time revealed a diffusely swollen lower lip, with a hard, nodular mass extending from side to side, deep in the lip tissue. The mucous membrane was fissured at the center of the lip. The surrounding areas had the characteristic induration which we have found in carcinoma cases. Because the lesion seemed to be malignant, a biopsy was taken and immediate irradiation administered. A wax mold containing 6—5 mg. tubes of radium was applied to the lip and a dosage of 2160 mg. hours given over a period of three days. (The pathological report at this time showed a chronic, productive inflammatory lesion with definite epithelioid tubercles.) The condition was partially improved followed by a plastic operation producing an excellent result. The patient has remained well to date. (Fig. 1A and B.)

FIG. 1A. FIG. 1B.

FIG. 1A. Case 1. Edematous lip with fissure through center. Fissure was of a purplish-grayish color.
FIG. 1B. Case 1, after radium therapy and plastic operation. Lesion completely healed.

CASE II. J. B., forty-eight-year old laborer, was referred to the clinic as a case of malignancy of the mouth. He gave a history of a rapidly growing sore in the right corner of the mouth. The lesion began as a small ulcer six months previously and had been growing rapidly. The patient gave a history of a chronic cough for the past eleven years, though his general condition seemingly was excellent. He had not lost weight and had been working steadily. Examination of the mouth showed an ulcer involving the entire angle of the mouth on the right side, extending out on the skin for about 0.5 cm. There was a good deal of edema of the surrounding tissues, and, in addition, there was a small, reddened area on the hard palate. Tuberculosis was suspected, which was confirmed by biopsy, which showed miliary tuberculosis; and an *x*-ray of the chest showed cavitation in both apices. Because of the pulmonary disease the patient was referred to the tuberculosis service for treatment. (Fig. 2.)

FIG. 2. Case II. Six month ulcer with surrounding edema and numerous fissures. History and *x*-ray were positive for pulmonary tuberculosis.

CASE III. J. S., a city fireman, thirty-one years of age, gave a history of a sore mouth of six months' duration. He gave no history suggestive of tuberculosis, except for a cough of three weeks' duration. Examination revealed that the entire buccal mucosa on the right side was studded with small yellowish granules. There was a superficial crusting of the skin at the right corner of the mouth. In addition, the entire right side of the face was markedly swollen. (Fig. 3.) *X*-ray of the chest showed signs of extensive tuberculosis. While this case was clinically the most characteristic of the group, several biopsies failed to show histological signs of tuberculosis. The patient was referred to the tuberculosis service for treatment, but because of the severity of his condition, death rapidly ensued.

CASE IV. A. H., a forty-nine-year old tailor, gave a history of an ulceration in the mouth of three months' duration. Examination revealed on the left buccal mucous membrane a raised area, about 2 inches in diameter, grayish-white in color, and fissured throughout. It was moderately tender, but firm, and the surrounding area was moderately swollen. There were no bleeding areas and no palpable cervical glands. A provisional diagnosis of leucoplakia was

made and a biopsy taken. It was reported as a squamous-cell epithelioma. On this basis, radium needles were inserted into the lesion

Fig. 3. Case III. Edematous right cheek with superficial fissured ulceration. Cheek mucosa was similarly diffusely involved. Chest *x*-ray was positive for phthisis.

and a dose of 1150 mg. hours administered. The condition improved, but failed to clear up completely. Further radiation was given six months later but because the condition failed to heal completely, or extend further, or involve the regional glands, another biopsy was taken. This showed tuberculosis. Re-examination of the original slide demonstrated the previous error, for the original diagnosis, too, should have been tuberculosis. An *x*-ray of the chest showed evidences of tuberculosis and the patient was referred to the tuberculosis service where, under sanitorium treatment, he improved remarkably.

Case V. E. B., white male, aged thirty-two years, entered the clinic with a history of a sore on lip of two months' duration. He gave no history of pulmonary tuberculosis. Examination revealed a small, indurated ulcer, ½ inch in diameter, on the right side of the upper lip. The ulcer was covered by a brown scab and the surrounding area of the lip was thickened, but felt soft and unlike the hard induration characteristic of malignancy. The clinical appearance of the lesion, and the unusual location on the upper lip, suggested a benign condition. Examination of the microscopic section showed the condition to be tuberculosis. Radon needles were inserted into the lesion and a dosage of 700 mc. hours was given. The lesion healed completely, and at the last return visit to the clinic, six months later, the patient's condition was excellent.

Case VI. H. L., a twenty-five-year old girl, was referred for treatment of a small sore on lower lip of five months' duration. Tuberculosis was suspected although there was no history of pulmonary tuberculosis and confirmation by

Fig. 4. Case IV. Photomicrograph of buccal mucosa. Section shows numerous characteristically appearing tubercles.

pathological examination could not be obtained. She was treated with surface application of radium, a dosage of 552 mg. hours being given. The lesion healed, recurred six months later, was treated again, and again recurred. The patient developed paralysis of the fifth, seventh, and eighth nerves and was referred to the neurological service. She went rapidly downhill, and died a year later. Autopsy showed tuberculosis with extension to the central nervous system.

Case VII. E. T., male, forty years of age, with a history of pulmonary tuberculosis of known duration of one year, reported to the clinic with a small ulcer on the left side of the tongue of six weeks' duration. The lesion was about 0.5 cm. in diameter and rested against a broken, jagged tooth, and was soft, not indurated. It was excised with the endotherm under local anesthesia. Pathological report of the specimen was tuberculosis. Subsequent history is lacking for the patient failed to return for follow-up examinations.

Case VIII. J. M., thirty-five-year old man, was referred to the clinic for treatment of a fissure on the tongue. Examination revealed a fissure on the dorsum of the tongue, about 3 cm. long and 1 cm. deep. The edges were slightly thickened but there was no induration present. The patient's mother and brother had tuberculosis, and he, himself, had also been treated for pulmonary tuberculosis for the past three years. Radium, in the form of platinum tubes, was applied to the lesion on the tongue. Four applications of 100 mg. hours each were given over a period of two months. The patient failed to return for follow-up examination.

Case IX. J. S., forty-two years of age, white, male, was referred from the Dental Clinic for treatment of an ulcer of the gum. The patient had had several teeth extracted six months previously and the present lesion had developed at the site of the extractions. Examination revealed an ulceration of the gum, 5 cm. by 3 cm. with a sharp edge, not hard nor indurated, but quite tender. A biopsy was taken and microscopic examination of the specimen showed a collection of epithelioid cells and occasional multinucleated giant cells resembling tubercles. *x*-ray examination of the chest revealed extensive nodular infiltration of the lungs. Three years previously the patient had had a tuberculous kidney removed. No radium treatment was given for the mouth lesion. He was referred to the tuberculosis service but his general condition as well as the local lesion in the mouth became progressively worse.

Case X. L. S., a twenty-seven-year old housewife, with a long-standing history of pulmonary and laryngeal tuberculosis, was referred for treatment of an ulcer of the pharynx. Examination revealed a small shallow ulcer on the posterior pharyngeal wall, about 1 cm. in diameter, with fairly well-defined edges, but no induration. The patient's general condition was very poor and she died before any radiation could be given.

SUMMARY

1. Tuberculosis of the mouth should be considered as a possible diagnosis in cases of chronic ulceration of the mouth.

2. While always secondary to pulmonary tuberculosis, the lung lesion may give few, or no, symptoms. It is in this group of cases in which errors in diagnosis are most likely to occur.

3. Radiation therapy may be of some value in the treatment of these cases.

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INJECTION OF ALCOHOL IN THE SPINAL CANAL FOR RELIEF OF PAIN*

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PAIN is the most common complaint of patients and they come to the doctor for its relief. In many conditions, especially those of acute onset, the proper treatment of the disease results in a subsequent cure and an accompanying subsidence of the pain. On the other hand, there are large numbers of patients with chronic diseases, both benign and malignant, whom we cannot hope to cure. These helpless individuals, whom it is impossible to cure, merit at least relief from pain. Too often these chronic incurables, especially those with inoperable malignancies, are neglected and soon drift to the quacks and cults for treatment. A little more care and consideration applied to make them more comfortable would retain their confidence in scientific medicine and make their final months and days less painful to themselves and less anguishing to family and friends.

In the care of this group of cases Dr. Dogliotti[1] reported a very unique and valuable procedure, a method of relieving pain by injection of absolute alcohol in the spinal canal. The purpose of this method is to reach the posterior roots as they emerge from the medulla and partially to reduce the sensitiveness of the region which is the seat of pain. It has been proved experimentally that alcohol floats for a long time on the cerebrospinal fluid and that it causes partial analgesia, without exciting motor disturbances or producing meningeal irritation. The technique as described by Dogliotti is as follows:

Introduce a lumbar puncture needle into the subarachnoid space, at the level of the roots most involved by the pain. Stretch the patient out on the side opposite to the painful side, in decubitus. Slowly inject 0.2–0.4 c.c. of absolute alcohol into the cerebrospinal fluid. The alcohol, due to its low density, penetrates up to the line of spinal roots immediately suprajacent to its point of introduction. By raising the pelvis from 10 to 20°, the alcohol reaches the sacral roots. If the trunk is elevated, it turns toward the thoracic roots. The patient has a sensation of heat and slight burning in the regions corresponding to the roots involved and only on the side opposite that which rests on the bed.

After five to ten minutes, there is produced a hypo-esthesia in the corresponding region, and the pain disappears. In a few cases, one observes a very slight muscular hypotonia. At the end of a few hours, in 50 per cent of the cases, the pain reappears with a headache which lasts from one to four days. At the end of this period, the patient notices a progressive improvement, and at the end of five to fifteen days he is more or less completely cured. After a few days, sensitivity is normal and there remain only a few small zones of hypo-esthesia.

I have followed this technique except as to size of dose, using from 0.5–1 c.c. I have also kept the patient on the side for two hours as suggested by Yeomans. In 2 cases, with bilateral pain, I repeated the injection ten days later with the patient on the opposite side. In no case have I seen any permanent involvement of motor nerves nor has there been any disturbance of the sphincters. Superficial sensory responses to heat, cold, touch

[1] DOGLIOTTI, A. M. Nouvelle méthode therapeutique pour les algies peripheriques: Injection d'alcool dans l'espace sous arachnoidien. *Rev. neurol.*, 2: 485, 1931.

* Read before the Los Angeles Surgical Society April 13, 1934.

etc., have been unchanged after the first few days.

Case I. This patient, a female, forty years of age, single, a graduate nurse, had a carcinoma of the right breast of one and one-half years' duration, and had refused operative procedure. When I first saw her in October, 1933, the condition was far advanced with a metastatic involvement of the right leg about the knee. She was suffering intensely, particularly with the leg, and was taking morphine and dilaudid hypodermically in large doses; ½ grain of morphine or 1/16 grain of dilaudid every three or four hours. This relieved the pain in the breast but had very little effect on the pain in the leg. The leg was so sensitive that she could not bear the weight of the bed-clothes on it, and would cry out if the knee was merely touched. This patient was placed on her left side, a spinal puncture made between the second and third lumbar vertebra and 1 c.c. of 95 per cent alcohol slowly injected. She immediately experienced a sensation of warmth along the leg and into the breast. The following day there was much less pain and there was a slight muscular weakness in the right leg. The next day she was able to walk to the bath room without causing any discomfort to the leg which forty-eight hours before one could barely touch because of the severe pain.

The opiates were gradually withdrawn, and in four or five days she was taking only 1/32 or 1/16 grains of dilaudid and was sleeping the whole night through on a capsule of amytol compound. This patient lived for the succeeding three months with comparative freedom from pain, during which time the malignancy progressed and involved the opposite breast and the skin over a large area of the chest. Throughout this period she had practically no pain and sedatives were required only for the nervous condition. Sensation for heat and cold were present and there was no motor disturbance in the leg except for slight weakness the first day.

Case II. Briefly, this patient, a young female adult, had a bacterial endocarditis, and as a complication a large aneurysm of the left femoral artery directly below Poupart's ligament. I shall not go into the details of this case except to say that this large aneurysm, which made a mass 6 or 8 inches in diameter, was extremely painful, and the patient was taking large amounts of opiate. One cubic centimeter of absolute alcohol was injected into the spinal canal. Within forty-eight hours her pain was relieved so that the leg could be moved with ease, and the aneurysm could be palpated firmly without complaint of pain. This patient died two weeks later from a rupture of the aneurysm, but during that period she was free from pain, comfortable and happy.

Case III. This is a case of an elderly gentleman with inoperable carcinoma of the prostate, on whom a superpubic cystotomy was done ten months ago. For several months his pain had been severe enough to require four hypodermic injections of dilaudid, each 1/20 grain, during the night. Six months ago 1 c.c. of 95 per cent alcohol was injected into the spinal canal with the patient on the right side. Ten days later the injection was repeated with the patient on the left side, as it is necessary to reach the sensory nerves on both sides of the cord for bilateral pain. There was a slight muscular weakness in the left leg for three or four days following the first injection, but this entirely recovered. Since the second injection his opiate requirements have been reduced by half. Recent roentgenological examination reveals metastases in the right ilium and fourth and fifth lumbar vertebrae. In spite of this he eats and sleeps well, has maintained his bodily strength sufficiently to be up in a chair daily.

Case IV. Mrs. G., fifty-five years old, seen in consultation because of severe muscle contraction and pain in left foot, apparently resulting from diabetic endarteritis. Patient had been bedfast for several weeks because of pain, which was aggravated by use. Opiaes were, necessary for rest. Alcohol injection twas done and within two days patient had complete relief of pain in foot, although muscle spasm persisted. Two weeks later she developed severe pain in the left sacroiliac area and along the sciatic nerve. A second injection of alcohol was given and in forty-eight hours the pain subsided and there has been no recurrence either in the foot or hip. This lady now does all her housework and sleeps without sedatives.

These cases are typical of the 12 in my series, and the results verify those of Dogliotti, Yeomans,[2] and Saltzstein.[3] The number of reported cases so treated is sufficiently large to warrant the conclusion that this is an efficient method of controlling pain in certain chronic and incurable diseases.

[2] Yeomans, F. C. Care of advanced carcinoma of the gastrointestinal tract. *J. A. M. A.*, 101: 1141, 1933.

[3] Saltzstein, H. C. Intraspinal (subarachnoid) injection of absolute alcohol for the control of pain in far advanced malignant growths. *J. A. M. A.*, 103: No. 4, 1934.

CONGENITAL HEMOLYTIC ICTERUS WITH CHOLELITHIASIS

IN A BOY FOUR YEARS OLD CURED BY SPLENECTOMY AND CHOLECYSTOSTOMY

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H. F., white male, aged four years, was admitted to Harper Hospital February 5, 1934, complaining of jaundice of twelve hours' duration; anemia and enlarged spleen of four months' duration.

Present Illness. Mother states that patient was well until October 1933, when general onset of pallor began, with loss of appetite and general lassitude. He was seen by a physician who stated that he had an enlarged spleen with anemia (hemoglobin 20 per cent). On therapy consisting of iron, cod-liver oil, high vitamin diet, and ultraviolet light treatments over the entire body, with concentration over the spleen, the condition did not improve much. No hemorrhage from mouth, nose or rectum. No petechiae or ecchymoses. No hematuria, diarrhea or jaundice, no chills or fever. No polyuria or dysuria. No pain, nausea or vomiting. No swollen joints or tenderness over bones. On January 17, 1934 a blood transfusion was given, followed by a slight reaction with chill and elevation of temperature. Since this time he has been confined to bed with no complaints except anorexia and malaise. Not constipated. Twelve hours ago parents noticed a rapidly developing yellow tint to patient's skin. Child has been nauseated and vomited food twice, and has had some fever.

Past History: Child was full term, spontaneous delivery baby. Had icterus neonatorum for first week of life which gradually subsided. Breast fed for nine months. Development normal. Walked at fourteen months. Always bright and alert. Was fat and chubby, but has always been somewhat pale. Suffered from no infectious diseases, and enjoyed good health until present illness.

Family History. Father, mother and one sister living and well. No familial or hereditary history of jaundice or hemophilia. No tuberculosis, cancer, renal or cardiac disease, diabetes or epilepsy.

Physical Examination. Well developed and nourished child with a marked deep jaundice. Temperature 100.4°F. Pulse 100. Respirations 30. There are no skin eruptions or glandular enlargements. The skeleton and head appear normal. There are no petechiae. No tenderness or swelling over the long bones. Pupils are equal and react to light and accommodation. Sclera deeply jaundiced. Mucous membranes smooth, soft and moist. Tongue clean, teeth in good condition. Tonsils small. Nose and ears normal. Neck, heart and lungs showed no pathology. Abdomen is moderately distended but not tender. The liver edge is three finger-breadths below the costal margin, smooth and not tender. The spleen is markedly enlarged filling the entire upper left quadrant, with a moderate dome which elevates the left costal margin. The surface is smooth and the edge sharp. The extremities, reflexes, and rectal examination were normal.

Laboratory Findings. Blood examination: reticulocytes 19.2, color index 1.0 hemoglobin 42; red blood cells 2,150,000; mean diameter of red blood cells 7.2 μ. White blood cells 8050; neutrophiles: segmented 31, stab 32, juvenile 5 and lymphocytes 32. Icteric index 140. Fragility: beginning hemolysis 0.5 per cent, complete hemolysis 0.3 per cent. Platelet count 45,000.

Bleeding time ½ minute. Clotting time 4 minutes. Sugar 0.105 per cent, nitrogen 33.4 mg. Moderate anisocytosis and slight poikilocytosis. Some red blood cells with Howell-Jolly bodies and stippling. Marked polychromasia. The microcystosis is moderate. No erythroblasts observed. Left shift hyperregenerative-irritative. Mild toxic cytoplasmic changes (coarse granules). The thrombocytes are normal in size and staining. VandenBerg reaction: direct and indirect positive. Wassermann reaction: negative.

Urine: highly colored, acid, specific gravity 1.030, no albumin, sugar or acetone. Bile 4 plus. Sediment negative.

X-ray of skull showed no pathology.

Preoperative Course. The child was given *x*-ray treatments over the spleen on the day of admission and three days later. Daily intramuscular treatments of liver extract were given. The icterus cleared up very rapidly and the condition of the patient markedly improved. The spleen however remained large, although a slight reduction may have taken place. The jaundice gradually disappeared, and at the end of the first week of hospitalization the icteric index was 3, the VandenBerg reaction was direct and indirect negative and the blood count was as follows: reticulocytes 33.2, color index 0.9, hemoglobin 60, red blood cells 3,105,000, white blood cells 15,020, neutrophiles: segmented 24, stab 40, juvenile 5, myelocytes 2, lymphocytes 22, monocytes 3, eosinophiles 3, basophiles 1, and platelets 472,300.

The patient continued to improve and we decided to do a splenectomy, when suddenly on February 20, 1934, fifteen days after admission, he became intensely jaundiced again, this coming on within a few hours. The third *x*-ray treatment was given over the spleen and the jaundice gradually disappeared in the next three days. Splenectomy was planned for February 24, 1934 and one hour before the operation he was given 230 c.c. of whole blood, using his father as donor.

Operation. Nitrous oxide ether anesthesia. Left rectus incision. Large spleen presented itself into wound, color slightly grayish with smooth surface. Liver enlarged with definite evidence of hepatitis. Few adhesions around gall bladder, which was dark colored, moderately distended and thickened. Stones were palpated in the gall bladder. Gall bladder opened, walls edematous, contained very thick bile and many small stones, which were removed. The gall bladder was drained and a moderate sized appendix was quickly removed. The spleen was immobilized and easily removed, and the wound closed, the patient leaving the operating room in very good condition.

Pathological examination of spleen showed fibrosis, congestion and moderate myolysis. Weight 437 gm.

Postoperative Course. Uneventful recovery from operation. Was kept in oxygen tent for twenty-four hours. Given 3200 c.c. saline by hypodermoclysis at the rate of 100 c.c. per hour. Fluids by mouth till the fourth day, then gradually increasing high caloric diet. Gall bladder tube came out spontaneously on the eleventh day. General condition and color gradually improved and patient was discharged from the hospital on March 23, 1934. The blood count before discharge showed reticulocytes 3.7, color index 0.9, R. B. C., 3,800,000, W. B. C. 12,250, platelets 432,000, differential count: neutrophiles: segmented 37, stab 15, juveniles 5, lymphocytes 33, monocytes 6, eosinophiles 3, basophiles 1. A few red cells showed Jolly bodies. The blood count showed a normal picture one month after leaving the hospital and has been normal since. The child is now the picture of health.

CONCLUSIONS

1. An interesting case of congenital hemolytic jaundice, associated with splenomegaly, splenic anemia, cholecystitis and cholelithiasis, is reported.

2. The gratifying result in this case is due to the rational medical treatment before operation, with the full cooperation of the *x*-ray therapist.

3. We believe that in every operation where splenectomy is contemplated, a thorough examination of the biliary tract is essential, and drainage of the gall bladder is indicated more often than usually practiced.

NEW INSTRUMENTS

NEW INTRA-UTERINE ELECTROCAUTERY

FOR THE DICKINSON METHOD OF PERMANENT STERILIZATION OF THE FEMALE

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IN 1916 Dickinson reported the use of an electrocautery to produce burns at the tubo-uterine junction, which, when followed by scar tissue contractions, blocked the tubal lumen and permanently sterilized the patient.[1] This was done under local anesthesia as an office procedure. In following his technique, it seemed more effective to use a different intra-uterine cautery, and to check the results of the operation with the injection of iodized oil into the uterine cavity.

FIG. 1.

The cautery is made in the form of a uterine sound with a small coil of platinum wire at its tip. It can be heated with an electric current from any rheostat. The terminal portion of the cautery is smaller than the shaft and is made flexible so that it may be bent to the shape most desirable in locating and cauterizing the uterine cornua. The instrument is graduated in inches and a slip collar allows the depth of insertion to be definitely limited. There is a projection at the hub that allows one to determine the position of the tip while in the uterus. The entire instrument may be sterilized in an antiseptic solution and because of its smooth surface is easily cleaned.

This method of sterilization should only be used in patients who have normal uteri. Pelvic infections will cause a stormy reaction and small submucous fibroids will make location of the tubal openings difficult. The best time for sterilization is during or immediately after a menstrual period.

The technique is as follows: The patient is placed in a lithotomy position and the perineum and vagina prepared as for any vaginal operation. Either under local anesthesia as suggested by Dickinson, or under intravenous evipal anesthesia, the cervix is exposed and grasped with a tenaculum. A graduated uterine sound is introduced into the uterine cavity to determine its shape and size. The tubal openings are located by the sense of touch. There is a definite resistance to traction in any other direction when the tubal opening is touched because of the funnel shape of the uterine cavity in this region. The depth of the two regions is noted on the graduations of the sound. The cautery

[1] Dickinson, R. L. *Surg. Gynec. Obst*, 23: 203, 1916.

is then bent into the same shape as the sound and its collar placed so that the depth noted in the sound cannot be exceeded. The cautery is heated to a cherry red and the tip buried in the anterior lip of the cervix or the lateral vaginal wall, with very slight pressure. The duration of time necessary to bury the tip is noted. The burn must be in a location easily visible with a bivalve speculum. The cautery is then withdrawn and cooled. It is next inserted in the uterine cavity and the tubal opening located as with the sound. The same amount of current for the same time is used as before. The cautery is cooled, withdrawn and re-inserted on the opposite side and the process repeated. The entire operation should take no more than ten minutes.

The vagina is kept clean and the burn in the vagina inspected at frequent intervals. When it is healed, it is presumed that the tubal openings are similarly healed. After the next menstrual period, the uterine cavity is then injected with iodized oil under fluoroscopic control by means of the Jarcho or similar apparatus. If no oil escapes into the tubes, the openings are strictured and the patient is sterile.

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[Concluded on p. 325.]

* Continued from p. 243.

SLING-SUPPORT FOR USE IN INJURIES OF THE UPPER EXTREMITY*

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MANY patients forced to wear a sling because of an injury to the upper extremity, complain of the limitation of motion, stiffness, and cramping of the neck which the use of such a support entails. Any increase in weight such as results from encasing the extremity in plaster or splints serves to further discomfit these patients. Several years ago, Dr. Donald Gordon of New York so modified the sling as to obviate these objectionable features. The modification consisted of attaching a one-yard strip of bandage to the two tails of the sling. The strips are carried over the shoulders, crossed behind the back, brought about the waist, and tied in front. In this manner the load is transmitted evenly to both shoulders and allows free motion of the neck. Although the method is effective it is not entirely satisfactory because of the failure of the strips to lie flat, and their tendency to slip upwards, to cause discomfort in warm weather, and to roll up and cause chafing under the breasts in females. In an attempt to overcome these difficulties, the author devised a light, durable apparatus which in practice has proved highly satisfactory. It can be readily made at home.

The device is constructed throughout of light, non-elastic, webbed material 1¼–1½ inch in width, and is comprised of two parts. (Fig. 1.) The first part consists of a belt (AC) divided at its mid-point by a metal ring (B) 3 inches in diameter. When the belt is buckled about the waist, the ring (B) lies in the midline over the vertebral column. The second part of the apparatus consists of a strip (DE) with a buckle attached to each end. This strip at its midpoint passes through the metal ring (B) of the belt (AC) previously described, while the ends of the former with their respective buckles pass upward, one toward each shoulder. (Figure 1 shows the apparatus assembled.) The injured arm is placed in a sling of conventional type, the tails of which are then fastened into the buckles at the ends of the shoulder strap (DE). It will be seen from Figures 2 and 3 that when employing this device, no strain is placed on the back of the neck and that the free mobility of the latter is unimpaired.

FIG. 1. Apparatus assembled. Belt A-C goes about waist. Strip D-E passes through ring B, and extends upward to shoulders. Note buckles at points D and E, seen indistinctly in photograph.

In the event of a complicating injury to one of the shoulders or clavicles, which

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often renders the wearing of the usual type of sling painful, both tails of the sling may be carried over the uninjured shoulder. and then across the front of the sling instead of behind it as is customary. The buckled belt now holds the sling firmly

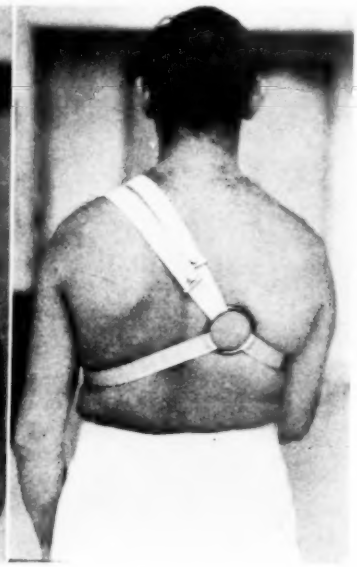

Fig. 2. Fig. 3. Fig. 4.

Fig. 2. Apparatus in use (anterior view). Conventional sling is employed. Tails of sling are attached to buckles at end of strip D-E.

Fig. 3. Apparatus in use (posterior view). Note that neck is entirely free of any strain.

Fig. 4. Injury to right arm and right shoulder. Entire weight is supported by left shoulder. Note rotation of belt so that ring B lies to opposite side of midline. This prevents strip D-E from slipping off shoulder.

(Fig. 4.) The injured side is thus completely spared. Under such circumstances, the strips are prevented from slipping off the shoulder by rotating the belt about the trunk until the metal ring (B) lies to the side of the midline of the back, away from the shoulder which supports the sling. (Fig. 4.)

In use, the apparatus is applied by first buckling the belt (AC) loosely about the trunk, just below the level of the costal arch. The latter tends to hold the belt in place and prevents it from later rising into the axillae. The shoulder strap (DE) is then passed through the ring (B), and its length adjusted until the buckles which receive the tails of the sling lie approximately over the front of the shoulders. (Fig. 2.) The shoulder strap (DE) may also be varied in length according to the amount of flexion of the forearm that is desired.

If more complete fixation of the upper extremity is desired, the belt (AC), before being buckled, is passed behind the elbow against the body. This variation may be employed regardless of whether the tails of the sling pass over one or over both shoulders.

Because of its light construction the support can be worn with comfort in warm weather and either over or beneath the clothing. While its use may not be necessary in every case, in many instances in which elderly or hypersensitive individuals are concerned, it affords a degree of comfort for which the patient is exceedingly grateful. In addition, in cases in which a complicating injury to the shoulder or clavicle makes the wearing of the conventional type of sling intolerable, it has been found at times to be indispensable.

The device may also be used as a corrective in cases of round-shoulder, and to replace the well known figure-of-eight dressing in the treatment of certain types of fracture of the clavicle. When employed for these purposes, it is applied in the usual manner. A 12 inch length of heavy, unbleached muslin, well padded with cotton,

is looped over the front of each shoulder and the two free ends on each side are then fastened into their respective buckles at the ends of the shoulder strap (DE). The latter is then shortened until the shoulders are drawn well back into the desired position.

The details of construction are shown in Figure 1. The following material, purchaseable at small cost, is required:

One strip of non-elastic webbed material or light canvas 1¼ to 1½ inches wide and 48 inches long. Two similar strips each 20 inches long. Three buckles, to fit the above. Three belt-shortening clips, to fit the buckles. One metal ring 3 inches in diameter of light weight.

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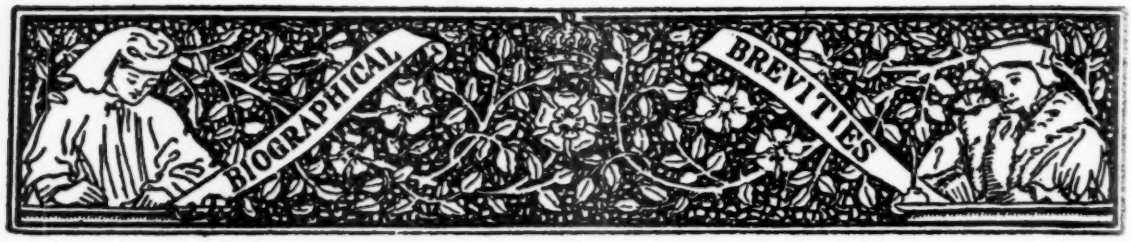
* Continued from p. 322.



CHARLES CALDWELL

CHARLES CALDWELL, the youngest of a large family, was born of pure Hibernian blood, in Caswell County, North Carolina, on May 14, 1782.

His was a turbulent life. A natural orator, a man of forceful personality, all his life he went from one controversy to another with his medical colleagues, and his years were marked by an accumulation of distinguished enemies. In his time, he founded two schools in the west, and wrote an "Autobiography" (1855) which was a remarkable diatribe, surcharged with venom and rancor, but which we are told "remains a storehouse of facts (and fancies) relating to the University of Pennsylvania, to Rush and to the early days of the Transylvania University and the Cincinnati schools. Pickled in vinegar, the work is sure to survive."

His early schooling was meagre except that his teacher of Latin, Dominie Harris, awoke oratorical aspirations in his fertile mind. At the age of fourteen, his parents died and Caldwell took charge of Snow Creek Seminary in North Carolina, and at the age of eighteen was called to establish another preparatory school in that state.

Prior to studying medicine, he studied under a Dr. Harris of Salisbury, N. C., and referred to this as a waste of time. He completed his first course of lectures at Pennsylvania in 1793. That fall a terrible epidemic of yellow fever broke out in Philadelphia. "His [Caldwell's] conduct in the pestilence . . . would assure him a place of honor in American Medicine." He fell out with Dr. Rush over the treatment of yellow fever and omitted any mention of it in his inaugural thesis (1796).

Caldwell helped found the Academy of Medicine and served as vice-president for two terms (1798–99). Later on he was dropped for non-payment of dues. He belongs to that group of American physicians who early put vaccination to "the practical test of personal application." In 1803 he introduced clinical instruction into Blockley (an alms-house).

He was offered a professorship in the new school at Greenfield, N. Y. and later a chair in Baltimore. At this time of his life he was drifting from one acrimonious controversy to another.

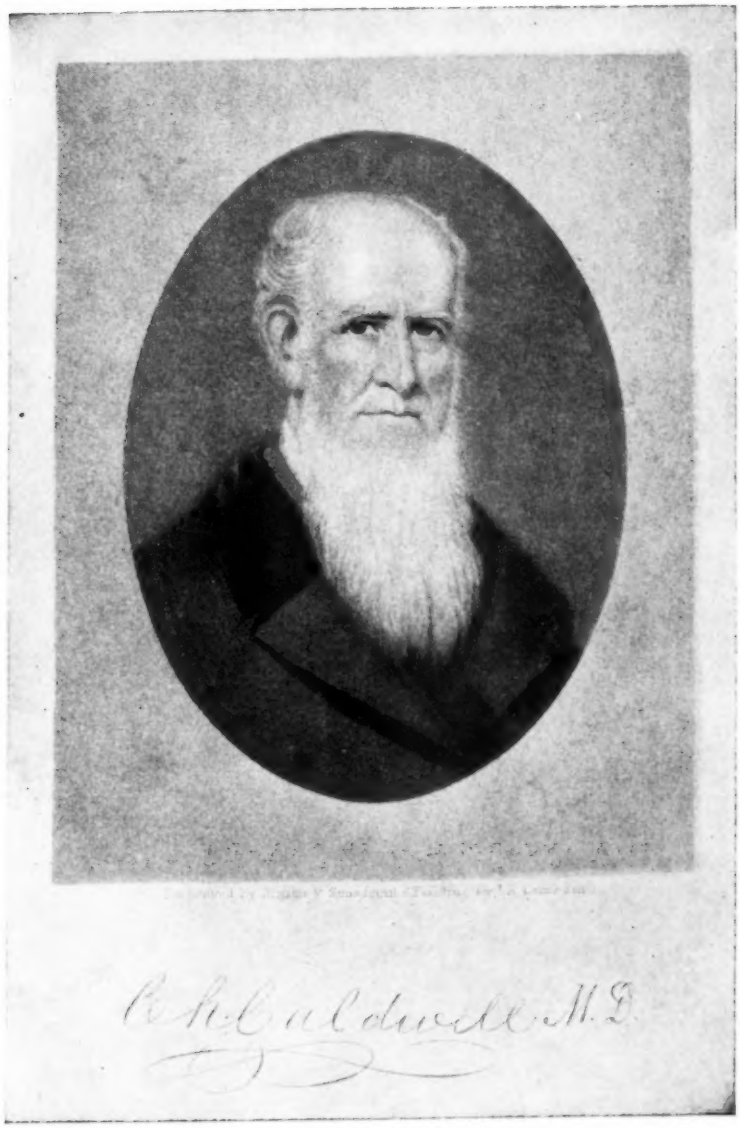
Caldwell did some important translations from foreign works and was editor of the *Port Folio*, a journal of national prominence during the War of 1812. In 1819 he wrote "Life and Campaigns of General Greene." In 1810 he started a course of lectures on medical jurisprudence.

His suggestion of a medical department at Transylvania University was approved by the trustees, and he was called to the chair of the institutes of medicine and clinical practice in August, 1819. Later, he engineered a movement to transplant the medical faculty from Transylvania to Louisville. He was made the scapegoat of this intrigue and was officially dismissed from the faculty. Undaunted he went among the people and raised funds for starting another school.

Little is known of his family life. He married twice. By his first wife, whom he divorced, he had a son who attended Harvard.

In May, 1853, this old war-horse contracted erysipelas. He never recovered and died on June 9, 1853. He was buried in Cave Hill Cemetery, Louisville, Ky.

T. S. W.



CHARLES CALDWELL

[1782-1853]

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

SIR PERCIVALL POTT—SURGEON ST. BARTHOLOMEW'S HOSPITAL

FELIX CUNHA, M.D.

SAN FRANCISCO, CALIF.

TO read the original treatise of Sir Percivall Pott describing the disease which today bears his name is exceptionally interesting because of the many sidelights it throws on the manner of his reasoning as to etiology, as to method of treatment and how this reasoning was arrived at. The number of cures attested to is also interesting, although we would be inclined to accept them with a bit of reserve. Then also the fact that attention was drawn to the disease or the diagnosis only after paralysis from cord pressure due either to destruction of vertebrae or impingement of the cord had set in.

As can be seen from the title page of the treatise (Fig. 1) the malady is described as "That kind of palsy of the Lower Limbs which is frequently found to accompany a Curvature of the Spine."

As the entire treatise is short, well worth reading and as copies of the original are scarce, I am quoting the entire article.

Among the various objects of Physick and Surgery, there are unfortunately some in which all the efforts of both, have hitherto been found absolutely ineffectual, and which therefore have always made a very disagreeable and melancholy part of practice.

To remove, or even to relieve any of the miseries to which mankind are liable, is a very satisfactory employment; but to attend on a distemper from its beginning, through a long and painful course, to its last, fatal period, without even the hope of being able to do anything which shall be really serviceable, is of all tasks the most unpleasant.

In such cases, any attempts, however hazardous, provided they were rational, would be justifiable, certainly then, whatever is not in itself dangerous and affords the smallest ray of hope, ought to be embraced. I publish therefore an account of the good success which has attended a particular method of treating a disease, which has hitherto foiled all the efforts of art; and I do it with the principle that by inducing others to make the same experiment, I propagate the benefit.

The disease of which I mean to speak is generally called a palsy, as it consists in a total or partial abolition of the power of using and sometimes of even moving the lower limbs, in consequence, as is generally supposed, of a curvature of some part of the spine.

To this distemper both sexes and all ages are equally liable. If the patient be an infant, it becomes an object of constant though unavailing distress to its parents; if an adult, he is rendered perfectly helpless to himself, and useless to all others, which, of all possible states, is surely the very worst.

When this disease attacks an infant of only a year or two old, or under, the true cause of it is seldom discovered until some time after the effect has taken place, at least not by parents and nurses, who know not where to look for it. The child is said to be uncommonly backward in the use of his legs, or it is thought to have received some hurt in its birth.

When it affects a child who is old enough to have already walked, and who has been able to walk, the loss of the use of his legs is gradual, though in general not very slow. He at first complains of being soon tired, is languid, listless, and unwilling to move much, or at all briskly; in no great length of time after this he may be observed frequently to trip, and stumble, although there be no impediment in his way; and whenever he attempts to move briskly, he finds that his legs involuntarily cross each other, by which he is frequently thrown down, and that without stumbling; upon endeavoring to stand still and erect, without support, even for a few minutes, his knees give way and bend forward. When the distemper is a little farther advanced it will be found, that he cannot, without much difficulty and deliberation, direct either of his feet precisely to any exact point; and very soon after this, both thighs and legs lose a good deal of their natural sensibility, and become perfectly useless for all the purposes of locomotion. When an adult is the patient the progress of the distemper is much the same but rather quicker.

Until the curvature of the spine has been discovered, it generally passes for a nervous complaint; but when the state of the backbone has been adverted to, recourse is almost always had to some previous violence to account for it, some pulling, lifting, carrying, or drawing a heavy body, which is supposed to have hurt the back. In some few instances, this exertion may have been such as might be allowed to have been equal to the effect, but, in by much the majority, this is so far from being the case, that if it be admitted to have had any share at all in it, some predisposing cause, at least, must be looked for, in which, (in my opinion) consists the very essence of the disease.

I have, in compliance with custom, called the disease a palsy; but it should be observed that notwithstanding the lower limbs be rendered almost, or totally useless, yet there are some essential circumstances in which this affection differs from a common nervous palsy; the legs and thighs are, I have just said, rendered unfit for all the purposes of locomotion, and do also lose much of their natural sensibility, but notwithstanding this, they have neither the flabby feel, which a truly paralytic limb has, nor have they that seeming looseness at the joints, nor that total incapacity of resistance, which allows the latter to be twisted in almost all directions; on the contrary the joints have frequently a considerable degree of stiffness, particularly the ankles, by which stiffness the feet of children are generally pointed downward, and they are prevented from setting them flat upon the ground.

The curvature of the spine, which is supposed to be the cause of this complaint, varies in situation, extent and degree, being either in the neck or back, and sometimes (although very seldom) in the upper part of the loins, sometimes comprehending two vertebrae only, sometimes three, or more, by which the extent of the curve becomes necessarily more or less; but whatever may be the number of vertebrae concerned, or whatever may be the degree or extent of the curvature, the lower limbs only feel the effect—at least I have never once seen the arms affected by it.

This effect is also different in different subjects: some are rendered totally and absolutely incapable of walking in any manner, or with any help and that early in the course of the distemper; others can make a shift to move about with the help of crutches, or by grasping their own thighs with their hands; some can sit in an erect posture, or in a chair, without much trouble or fatigue, which others are incapable of, at least for any length of time; some have such a degree of motion in their legs and thighs, as to enable them to turn and move for their own convenience in bed, others have not that benefit, and are obliged to lie till moved by another.

When a naturally weak infant is the subject, and the curvature is in the vertebrae of the back, it is not infrequently productive of additional deformity, by gradually rendering the whole back what is commonly called humped; and by alterations which all the bones of the thorax sometimes undergo, in consequence of the flexure and weakness of the spine, by which such persons are justly said to be shortened in their stature; but in all cases where this effect has been gradually produced,

to whatever degree the deformity may extend, or however the alteration made in the disposition of the ribs and sternum may contribute to

REMARKS

ON THAT KIND OF

PALSY

OF THE

LOWER LIMBS,

WHICH IS FREQUENTLY FOUND TO ACCOMPANY A CURVATURE OF THE SPINE,

AND IS SUPPOSED TO BE CAUSED BY IT.

TOGETHER WITH

ITS METHOD OF CURE.

TO WHICH ARE ADDED,

OBSERVATIONS on the NECESSITY and PROPRIETY of AMPUTATION,

IN CERTAIN CASES,

AND UNDER CERTAIN CIRCUMSTANCES.

BY PERCIVALL POTT, F. R. S.
And Surgeon to St. Bartholomew's Hospital.

Verumque eſt ad ipſam curandi rationem nihil plus conferre quam experientiam. CELSUS.

LONDON:

Printed for J. JOHNSON, No. 72, St. Paul's Church-Yard.
M,DCC,LXXIX.

FIG. 1. Title page from Pott's original description of the disease.

a variety of complaints, dies in an exhausted emaciated state; or, which is still worse, drags on a miserable existence, confined to a great

FARTHER

REMARKS

ON THE

USELESS STATE

OF THE

LOWER LIMBS,

IN CONSEQUENCE OF A

CURVATURE OF THE SPINE:

BEING

A SUPPLEMENT to a former TREATISE on that Subject.

BY PERCIVALL POTT, F. R. S.
SURGEON TO ST. BARTHOLOMEW'S HOSPITAL.

LONDON:

Printed for J. JOHNSON, No. 72, St. Paul's Church Yard.
MDCCLXXXII.

FIG. 2. Title page from his second description published three years later.

such deformity, yet I think that it will always be found, that the curvature of the spine appeared first, and, if I may so say, singly and that all the rest was consequential.

While the curvature of the spine remains undiscovered or unattended to, the case is generally supposed to be nervous, and medicines so called are most frequently prescribed, together with warm liniments, embrocations and blisters, to the parts affected; and when the true cause is known, recourse is always had to steel stays, the swing, the screw chair, and other pieces of machinery, in order to restore the spine to its true and natural figure; but all, as far as I have observed, to no real or permanent good purpose; the patient becomes unhealthy, and languishing for some time under chair, or bed, totally deprived of the power of locomotion, and useless both to himself and others.

This in an infant is most melancholy to see, in an adult most miserable to endure.

The general health of the patient does not seem at first, to be materially, if at all, affected, but when the disease has been sometime, and the curvature thereby increased, many inconveniences and complaints come on, such as difficulty in respiration, indigestion, pain and what they call tightness at the stomach, obstinate constipations, purgings, involuntary flux of urine and faeces, etc., with the addition of what are called nervous complaints, some of which are caused by the alterations made in the form of the cavity of the thorax, others

seem to arise from impressions made on the abdominal viscera. These are different both in kind, and in degree, in different subjects, but seem to depend very much on the consequences of the curvature—that is, in naturally infirm children, although the curvature of the dorsal vertebrae is always the first mark of the distemper, by preceding every other, yet it is frequently soon followed by such a degree of deformity of the bones of the trunk, as to be, in conjunction with the necessary inactivity and confinement of the patient, productive of all the ills above mentioned.

An affecting instance of this distemper in the person of a very promising youth of fourteen years old, with whose family I was nearly connected, induced me to think more of it than perhaps I otherwise should have done; and the restoration of the use of his limbs, immediately after a seemingly accidental abscess near the part, engaged my attention still more, and became a matter of frequent, though not very satisfactory contemplation; I say unsatisfactory, because it served only to increase my doubts, without leading me toward a solution of them. The more I thought upon the subject, the more I was inclined to suspect that we had been misled by appearances, and that a distempered state of the parts forming, or in the neighborhood of curvature, preceded or accompanied it: in short, that there was something predisposing, and that we had most probably mistaken an effect for a cause.

For these suspicions, I had the following reasons which appeared to me to have some weight:

1. That I had never seen this paralytick effect on the legs from a mal-formation of the spine, however crooked such malformation might have rendered it, or whether such crookedness had been from time of birth, or had come on at any time afterwards during infancy.

2. That none of those strange twists and deviations, which the majority of European women get in their shapes, from the very absurd custom of dressing them in stays during their infancy, and which put them into all directions but the right, ever caused anything of this kind, however great the deformity might be.

3. That the curvature of the spine, which is accompanied by this affection of the limbs, whatever may be its degree, or extent, is at first almost always the same, that is, it is always from within, outward, and seldom or never to either side.

4. That since I had been particularly attentive to the disorder I had remarked, that neither the degree nor the extent of the curve, made any alteration in the nature or degree of the symptoms at first, nor for some time after the appearance; or in other words, that the smallest curvature, in which only two or three of the vertebrae were concerned, was always, at first, attended by the same symptoms as the largest.

5. That although it sometimes happened that a smart blow, or a violent strain had immediately preceded the appearance of the curve, and might be supposed to have given rise to it, yet in many more adults it happened that no such cause was fairly assignable, and that they began to stoop, and to falter in their walking, before they thought at all of their back, or of any violence offered to it.

6. That exactly the same symptoms are found in infants, and in young children, who have not exerted themselves nor have been injured by others, as in the adult, who has strained himself, or received a blow; and that the case was still the same in those grown people, who have neither done, nor suffered any act of violence.

7. That although it must be allowed, that a dislocation of any of the vertebrae, would most probably be attended with the same kind of symptoms from the pressure it must make on the spinal marrow, yet it is also most probable that such symptoms would be immediate, and attended with great pain in the part; neither of which is in general the case here.

These considerations appeared to me to have much force; but what confirmed me in my opinion was the state of the parts forming the curvature, and which I had several fair opportunities of examining after death. By those examinations I found in infants, in young children, and in those who had been affected with the disorder but a small space of time, that the ligaments connecting the vertebrae, which from a natural state, by being somewhat thickened and relaxed, and that what are called the bodies of those bones, were palpably spread and enlarged in their texture, just as the bones forming the articulations are in children who are called rickety. That those who had long labored under the distemper, and in whom the symptoms were aggravated, whatever might

be their age, the ligaments were still more thickened, relaxed, and altered, the bodies of the bones more spread, more enlarged, and more inclined to become carious, and the cartilages between the bodies of the vertebrae much compressed and lessened in size; and that in all those who had so long labored under the disease, as to have been destroyed by it or by its consequences, the corpora vertebrarium were completely carious, the intervening cartilages totally destroyed, and a quantity of fanies lodged between the rotten bones, and the membrane investing the spinal marrow.

All these circumstances put together, induced me, as I have already said, to suspect, that when we attribute the whole of this mischief to the mere accidental curvature of the spine, in consequence of violence, we mistake an effect for a cause, and that previous both to the paralytick state of the legs, and to the alteration of the figure of the backbone, there is a pre-disposing cause of both, consisting in a distempered state of the ligaments and bones, where the curve soon after makes its appearance.

While the subject was fresh in my mind, I happened to be at Worcester, and in a conversation on it with the late Doctor Cameron, of that place, I mentioned to him, my opinion, and my doubts; the Doctor concurred with me, and at the same time mentioned a circumstance, which made a strong impression on me. He said, that he remembered some years ago, to have noted a passage in Hippocrates, in which he speaks of a paralysis of the lower limbs being cured by an abscess in the back or loins, and that taking the hint from this, he, Doctor Cameron, had, in a case of palsy of the legs and thighs, attended by a curvature of the back bone, endeavored to imitate this act of nature, by exciting a discharge near the part, and that it had proved advantageous. He also referred me to Mr. Jeffrys, a surgeon of eminence at Worcester, for a farther account of the same kind of attempt; this gentleman confirmed what Doctor Cameron had told me and assured me that he had found the method equally successful.

It may easily be supposed, that these accounts from gentlemen of veracity, and of reputation in their profession, still added to my desire of knowing more on this subject, and determined me to lose no opportunity of getting information.

The first that offered was in an infant, whose curvature was in the middle of the neck, and who had lost the use of its legs for about two or three months. I made an issue by incision on one side of the projection, and gave strict charge to the mother to take care that the pea was kept in; the woman, who had no faith in the remedy, did not take the proper care, and consequently the discharge was not equal to what it should, and might have been, but notwithstanding this neglect, at the end of about three weeks or a month the child was manifestly better, and began to make use of its legs; it was then seized with the small pox and died. The bodies of the vertebrae concerned in the curve, were larger than they should be, and than those above and below were, and their texture much more open and spongy, which difference appeared immediately, before the parts covering them were dissected off.

Some time passed before I had another opportunity. My next patient was a tall thin man, about thirty-five years old, who thought that he had hurt himself by lifting a heavy weight: his legs and thighs were cold, and what he called nummy, but not useless: he could with difficulty go about the room with the help of a pair of crutches, but he could neither rise from his chair, nor get on his crutches without the assistance of another person, nor could he without them walk at all.

I made a seton on each side of the curve, which was in his back, about the middle, and having given his wife directions how to dress them, I called on him once in three or four days. At the end of six weeks he had recovered the due degree of sensation in his limbs, and found much less necessity for the use of his crutches; he could rise from his bed, and from his chair without assistance, and by means of one crutch, and an underhand stick, could walk for an hour, or more without resting, and without fatigue. The setons had now, from not having been properly managed, worn their way out, and I would have converted each of them into an issue, but as neither the patient nor his wife had believed that the discharge had had any share in his amendment, but on the contrary that he would have been better without it, he would not submit to what I proposed, and I left him. At the distance of about three weeks from the time of my leaving him, I met him on the street walking very stoutly, with a common cane, of which he made little or no use. I asked him

what he had done: he told me that the sores had continued to discharge till within a few days, but that he had drank a great deal of confrey-root tea, with isinglass, and he supposed that had cured him.

I believe that the cure of this man will, by all who know anything of medicine, be thought to be so unlikely to have been affected by the confrey and isinglass, that my influence in favour of the seton will not be thought unrea-

Fig. 3.

Fig. 4.

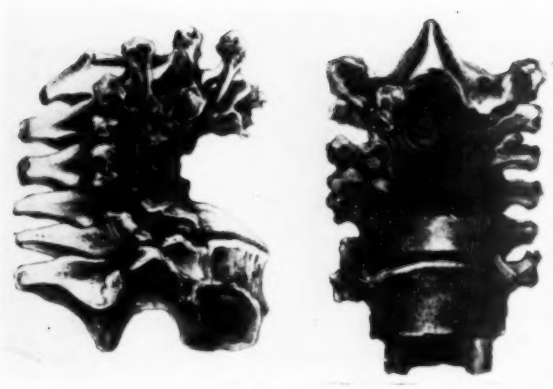


Fig. 5.

Fig. 6.

Fig. 3–6. Reproduced from Pott's own photographic plates of post-mortem material illustrating the extensive destructive changes described by him.

sonable, and that my determination to prosecute the method, from what I had heard and seen, was well founded.

Within the course of the last ten or twelve months, I have had several fair opportunities of doing this, both in St. Bartholomew's hospital, and out of it, and am very happy to be able to say, that it has not only always answered, but in some instances greatly exceeded my most sanguine expectations, by restoring several most miserable and totally helpless people to the use of their limbs, and to a capacity of enjoying life themselves, as well as of being useful to others.

I have now in the hospital a boy about twelve years old, whose case was so truly deplorable, that I made the experiment merely to avoid the appearance of inhumanity, by discharging him as incurable, without trying something. The curvature was in his back, and consisted of three or four vertebrae, but by means of the weakness thereby induced, the whole set of dorsal ones had so universally and gradually given way, that he was exceedingly deformed both behind and before: he was so absolutely incapable of motion, that he could neither turn himself, nor sit up in his bed: his feet were pointed downwards, and his ankles so stiff, that when he was held up under the arms, the extremities of his great toes touched the floor, nor could his feet be brought flat to the ground by any means, or force whatever. In short, he was as perfectly and as totally helpless as can be supposed, and at the same time in an exceeding general bad state of health, from the disorders of the thoracic and abdominal viscera. In this state he had been more than a year: it is now about three months since the caustics were applied; he is become healthy, and free from most of his general complaints, has the most perfect use of his legs while he is in bed, can walk without the assistance of any body, or anything to hold by; and from his manner of executing this, will, I make no doubt, in a very short space, recover perfectly the use of his legs. To this I ought to add, that notwithstanding a considerable degree of deformity does, and I suppose will, remain, yet the spine in general is so much strengthened, that he is some inches taller than he was four months ago.

The remedy for this most dreadful disease consists merely in procuring a large discharge of matter, by suppuration from underneath the membrana adiposa on each side of the curvature, and in maintaining such discharge until the patient shall have perfectly recovered the use of his legs.

To accomplish this purpose, I have made use of different means, such as setons, issues made by incision, and issues made by caustic; and although there be no very material difference, I do upon the whole prefer the last. A seton is a painful and nasty thing, beside which it frequently wears through the skin before the end for which it was made can be accomplished: issues made by if they be large enough for the intended purpose, are apt to become inflamed, and to be very troublesome before they come to suppuration; but openings made by caustic are not in general liable to any of these inconveniences, at least, not so frequently, nor in the same degree: they are neither so troublesome to make or to maintain. I make the eschars about this size and shape on each side the curve, taking care to leave a sufficient portion of skin between them; in a few days when the eschar begins to loosen and separate, I cut out all the middle, and put into each a large kidney-bean: when the bottoms of the sores are become clean by suppuration, I sprinkle every third or fourth day, a small quantity of finely powdered cantharides on them, by which the sores are prevented from contracting, the discharge increased, and possibly other benefit obtained. The issues I keep open until the cure is compleat, that is, until the patient recovers perfectly the use of his legs, or even for sometime longer, and I should think that it would be more prudent to heal only one of them first, keeping the other open for some time, that is, not only until the patient can walk, but until he can walk firmly, briskly, and without the assistance of a stick; until he can stand quite upright, and has recovered all the height, which the habit, or rather the necessity of stooping occasioned by the distemper, had made him lose.

I have said that the discharge by means of the issue is all that is requisite for a cure, which is true, as I have experimentally proved by not using any other in cases which have succeeded perfectly; but this fact being established, there is no reason why every assistant means should not be applied at the same time, in order to expedite: such as bark, cold-bathing, frictions, etc.

That the patient becomes more upright as his legs become stronger, is certain, and there-

fore appears taller as well as straighter in proportion, as the whole spine strengthens; but whether the curvature will always and totally disappear I am not yet able to say with certainty. In two late instances both adults, it has, but the deformity, which in weak infants and children, is often the consequence of the curvature, and of the state of the spine at that place, must in some degree, I fear, be expected to remain; but of this I am not yet able to speak with absolute certainty. There are a few other circumstances, of no great moment perhaps, but which will require more time to ascertain than I thought should be sufficed to pass, before mankind were made acquainted with the great means of relief, in so distressing, and so dreadful a malady: for the reader will be pleased to remember what I told him in the beginning of this tract, which was, that my motive for publishing this account sooner than might appear in general to be right, or indeed than I otherwise should have done, was a desire that as little time as possible might be lost, in conveying to the profession in particular, and to mankind in general, the means of relief under an affection, which till these were known, has not admitted of any: and this I was still more incited to do, because the remedy is as harmless, and as void of hazard, as it is efficacious.

And thus ends Sir Percivall Pott's description of a disease which apparently was recognized only when extensive vertebral destruction had taken place, with kyphosis—compression of the cord and paralysis—partial or complete of the legs. It is interesting to note that although mention is made of the frequent involvement of the thoracic viscera, association of that with the vertebral lesion was not thought of. A perusal of his plates shows that his only conception of the condition was in a very advanced state—when extensive bony destruction had taken place. Figures 3–6 illustrate this well.

However, his deductions, seven in number—differentiating this as a separate disease entity and not due to trauma or congenital defect are certainly complete and descriptive. His allusion to its presence in infants who had never had trauma of any sort to clinch his arguments.

One is at a loss, however, to explain his cures with their recovery of motion in the legs, disappearance of the deformity and attainment of former height. Local weakening of the structures about a cold abscess so that it might more readily rupture externally as he might have accomplished with his seton might account for a recovery of motion in the legs due to release of pressure upon the cord, but hardly the excellent results reported.

However due credit must be awarded Sir Percivall for recognizing the condition, taking it out of the realm of ignorance in which it had rested, actually looking at post-mortem material and describing it and at least suggesting some therapeutic procedure so that it is only befitting that it should bear his name as the pioneer in all this work.

His explanation and defense of his tract follows:

In the preceding tract I have related the appearance which the parts constituting the seat of the distemper make upon examination after death, or to speak more properly, the different states of these parts in different persons, and at different periods of this disease. These, though necessarily subject to considerable variety, may, I think, be reduced to three general ones.

I. A small degree of an increase of size in the bodies of the vertebrae, forming the curve, with an apparent laxity in their texture, and relaxed state of the connecting ligaments, by which they seem to have lost part of their power of holding the bones together.

II. A more considerable, and more apparent enlargement of the same parts of the vertebrae, whose spongy texture becomes more visibly spread through their whole substance, and tending towards a caries, with an apparently distempered state, both of the ligaments, and of the intervening cartilages.

III. A truly carious state of the bodies of the bones, a dissolution or destruction of the cartilaginous substance between them, and a lodgment of sanies on the surface of the membrane envelloping the spinal marrow.

These are I think the most particularly different states or stages of the disorder, and

are such as, in my opinion, decisively mark the true nature of it.

Between these in different persons, and under different circumstances, there must be a considerable variety, but the material difference will be only in degree.

From the whole, the few following practical inferences seem fairly deducible:

1. That the disease does not originally consist in a displacement of the vertebrae, made by violence, the bones and ligaments being previously in a sound and uninjured state; but in such a morbid alteration of the texture of both, as will, if not timely prevented, produce curvature and caries, with all their consequences.

2. That the proper remedies for this disease cannot be applied too soon.

3. That the restoration of the spine to its normal figure, depends much on the early administration of the help proposed.

4. That although the distemper may be so far cured, that the patient may perfectly recover the use of his legs, yet such an alteration may have taken place in the bodies of the vertebrae, as to render it impossible for the spine to become straight again.

5. That when three or four, or more vertebrae, are concerned in the curve, the trunk of the body will have so little support from that part of the spine which is not distempered, that no degree of deformity can be wondered at; nor can it be expected that such deformity should be removed, whatever other benefit such patient may receive.

6. That if from inattention, from length of time, or from any other circumstances, it happens that the bodies of the vertebrae become compleatly carious, and the intervening cartilages are destroyed, no assistance is to be expected from the proposed remedy.

To these I will take the liberty of adding that it appears to me well worth while, to try what a large and free discharge, made for a length of time from the vicinity of the distempered part, might be capable of doing in the very beginning of what are commonly called scrophulous joints; which when arrived to a certain point, baffle all our art, and render a painful and hazardous operation, absolutely necessary.

The description of the bone pathology was not strictly correct but from further study of postmortem material is made quite complete and detailed in treatise II published three years later. This shortcoming, however, is explained in his statement that he realized the paper a bit premature but that he considered the advancement of a simple remedy for a hopeless disease as sufficient justification.

In reading between the lines one can obtain a glimpse of the idealism of the man, such lines as "To remove, or even to relieve any one of the miseries to which mankind are liable, is a very satisfactory employment." Again, "I made the experiment merely to avoid the appearance of inhumanity."

And thus Sir Percivall Pott and the original description of the disease bearing his name.*

* From the author's collection of old medical books and medical incunabula.

BOOK REVIEWS

THE PRINCIPLES AND PRACTICE OF UROLOGY. By Frank Hinman, M.D. Phila. W. B. Saunders Co., 1935.

This new textbook of urology by Dr. Hinman presents the subject in a thorough, complete, up-to-date and yet concise fashion by one of the outstanding authorities in the country.

The point is made that the work is not written for the experienced urologist but for the student and general practitioner. "What they require," says the author, "is knowledge which will enable them to recognize a urological case when they see one." And it is believed that a thorough study of this book will place any competent practitioner in a position to do this.

The importance of general knowledge of the specialties today cannot be over-emphasized. At the same time, it must be remembered that important decisions and details of technique must be left to competent specialists. The presentation of Urology by Dr. Hinman might well be a model for books on other specialties for the general practitioner.

ECONOMIC PROBLEMS OF MEDICINE. By A. C. Christie, M.D. N. Y., Macmillan Co., 1935.

The very title of this volume is apt to tempt the reviewer into a long discussion of one of the most important phases of medical life today. The subject, of course, is capable of almost unlimited discussion. The author has succeeded in 12 chapters in presenting views which are those of a man who has studied his subject thoroughly, who is conservative and who is not afraid to express his sentiments. It is sure to take a definite place in the literature of the subject.

THE PRACTITIONERS LIBRARY OF MEDICINE AND SURGERY. Volume VIII, THERAPEUTICS, Supervising Editor, George Blumer, M.D. and Associate Editor, Albert J. Sullivan, M.D. N. Y., D. Appleton-Century Co., 1935.

This is a splendid resume of the all-important subject of therapeutics, covering treatment in every phase—from drugs through diet therapy to *x*-rays and physical therapy. The chapters are written by outstanding authors on each subject and written in a concise and understandable manner for the use of the general practitioner.

This volume is quite in keeping with the previous volumes of this set which is now nearing completion and promises to become one of the outstanding works of reference for the general practitioner.

BLOOD GROUPS AND BLOOD TRANSFUSION. By Alexander S. Wiener, M.D. Springfield, Ill., Charles C. Thomas, 1935.

The need for a standard book on this subject has long been felt and the author in a well written volume of two hundred pages has answered most of the questions regarding blood grouping that face the average practitioner. This book will find a niche in the library of everyone interested in the subject.

INTERNATIONAL CLINICS. Volume IV. Forty-Fourth Series, Edited by Louis Hamman, M.D. Phila., J. B. Lippincott Co., 1934.

Volume IV of the Forty-Fourth Series lives up to the standards set by the previous volumes. Selected articles covering special chapters on Medicine, Surgery, Obstetrics and Gynecology, Clinical Pathology, Recent Progress in Ophthalmology and Dermatology, the International Clinics, as usual, may be considered as supplementing all current textbooks on the subject.

TEXT-BOOK OF UROLOGY FOR STUDENTS AND PRACTITIONERS. By Daniel N. Eisendrath, M.D. and Harry C. Rolnick, M.D., Ed. 3, Rev. Phila., J. B. Lippincott Co., 1934.

This is the third edition, revised and brought up-to-date, the first edition having been published in 1928. Three editions in six years is in itself proof that this textbook on this so important subject has been a definite place for itself in medical literature.